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SYMPOSIUM ON THE LARYNX.

I.—PRACTICAL ANATOMICAL CONSIDERATIONS OF THE LARYNX.*

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The accurate anatomical description of the larynx is, to most laryngologists, a difficult thing to remember. While they undoubtedly could give a general anatomical description of it, still there would be many small but important omissions from it. This in no way reflects upon the laryngologists, but does indicate the complexity of this organ.

To the anatomist the study and conception of the larynx should and does mean one thing. But to the laryngologist this same organ, from its anatomical consideration, should and probably does convey a different impression.

The object of this presentation, therefore, is to set forth the laryngologist's anatomical conception of the larynx and its practical application.

Given the various structures which go to make up this important organ, suppose we attempt to reconstruct it. In other words, we shall present the plans and specifications of the larynx.

In the make-up of the larynx we have cartilages, bones, ligaments, membranes, muscles, nerves, blood vessels, lymphatics and mucous membranes.

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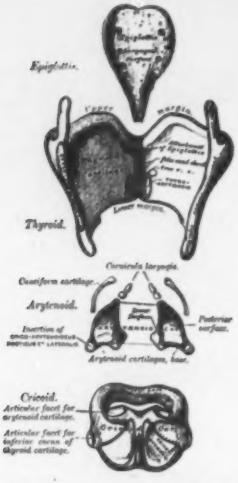

Fig. 1. The Cartilages of the Larynx. Posterior View (Gray's Anatomy).

This shows the separate cartilages, but they are not in the respective positions they occupy in the larynx.

Note particularly the muscular attachments.

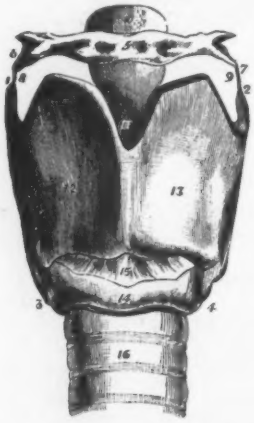

Fig. 2. Front View of the Larynx (Lennox Browne).

1, 2—Superior Cornua of Thyroid Cartilage. 3, 4—Inferior Cornua of Thyroid Cartilage. 5—Hyoid Bone. 6, 7—Cornua of Hyoid Bone. 8, 9—Thyrohyoid Ligaments. 10, 11—Epiglottis. 12, 13—Alae of Thyroid Cartilage. 14—Cricoid Cartilage. 15—Cricothyroid Membrane. 16—Trachea.

In this illustration the cartilages and hyoid bone are in their proper positions.

Bone and Cartilages: The rigid framework is of course made up of bone and cartilage. Rather than describe in detail, drawings will be used to set forth and refreshen your memory as to their shapes and relative positions.

You will note in Figs. 4, 5, 6 that the hyoid bone is included in the larynx. This is very important, as it is through it and its attached muscles that the larynx is suspended. Holding these rigid structures together we have ligaments and membranes. Of these, two are of particular importance—enough to warrant special mention and description.

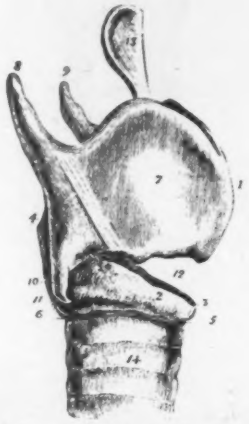

Fig. 3. Side View of the Larynx (Lennox Browne).
1—Prominence of Thyroid Cartilage (Pomum Adami). 2—Cricoid Cartilage. 3, 4—Upper Border of Cricoid. 5, 6—Lower Border of Cricoid. 7—Thyroid Cartilage. 8, 9—Superior Cornua of Thyroid. 10—Right Inferior Cornu of Thyroid. 11—Articulation of the Thyroid with the Cricoid. 12—Cricothyroid Aperture. 13—Epiglottis. 14—Trachea.
Here also the cartilages are in their proper positions.

Ligaments and Membranes: Bridging the gap between the upper border of the thyroid cartilage and the hyoid bone is the thyrohyoid membrane. "It is composed of fibrous tissue and extends in one continuous sheet from the upper border of the thyroid cartilage to the posterior surface of the hyoid bone. The posterior edges or borders which extend from the superior cornua of the thyroid cartilage to the tips of the greater cornua of the hyoid are thickened. They may be artificially dissected to resemble cords (ligamenta thyrohyoidea lateralis), although in fact they are continuous, not only

with the rest of the membrane, but with the expansion which mingles with the fascia of the neck. Quite often a nodule (cartilago

Fig. 4. Side View of the Larynx, Showing the Interior, the Right Plate of the Thyroid Being Removed (Lennox Browne).

1, 2—Arytenoid Cartilages. 3, 3—Processi Vocales of the Arytenoids. 4—Processus Musculus of the Right Arytenoid. 5—Upper Border of Cricoid. 6, 3, 3—Vocal Cords. 7—Facet for Articulation of the Thyroid with the Cricoid. 8—Left Plate of the Thyroid. 9—Left Superior Cornu of Thyroid. 10—Cricoid Cartilage. 11—Trachea.

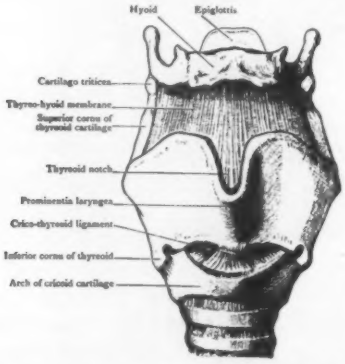

Fig. 5. Front View of Larynx, Showing Cartilages, Ligaments and Membranes (Cunningham's Anatomy).

Note particularly the thyrohyoid membrane and the cricothyroid ligament or conus elasticus.

triticea) is found in this lateral thickening. Deep to this membrane is a variable amount of fat which is bounded internally by the

mucous membrane lining the interior of the larynx. The space which this bridges is variable, it being greatest when the head is extended."[1]

The other, the cricothyroid membrane or conus elasticus, bridges the cricothyroid space.

According to Gray, the cricothyroid membrane and conus elasticus are identical. He states that "it consists of three parts, a central

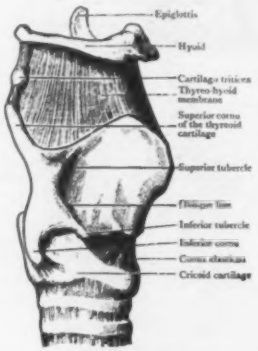

Fig. 6. Lateral View of Larynx, Showing Cartilages, Ligaments and Membranes (Cunningham's Anatomy).
Note also the extent of the thyrohyoid membrane and the conus elasticus.

triangular portion and two lateral portions. The central part (ligamentum cricothyreoideum medium) is thick and strong, narrow above and broadening out below. The lateral portions are thinner and lie close under the mucous membrane of the larynx. They extend from the superior border of the cricoid cartilage to the inferior margin of the true vocal cords with which they are continuous." Cunningham refers to the membrane as "the conus elasticus which is separable into a median and two lateral parts. The median part is the cricothyroid ligament. Each lateral part terminates in a free thickened border, called the ligamentum vocale."

This membrane or conus elasticus gains "attachments to the inner surface of the thyroid cartilage in front and to the body and vocal process of the arytenoid behind; therefore, it is correct to look upon the vocal cords as thickened portions of the conus elasticus."[2] We have now seen how the rigid framework of the larynx is bound together by the various ligaments and membranes (see Figs. 2, 5, 6, 7 and 8). Further binding these structures, we come to the various

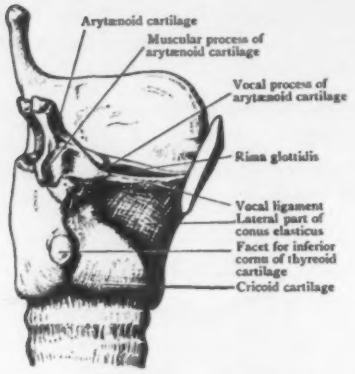

Fig. 7. Conus Elasticus. The Right Lamina of the Thyroid Cartilage Has Been Removed (Cunningham's Anatomy).

Note particularly the relationship of the conus elasticus to the vocal ligament.

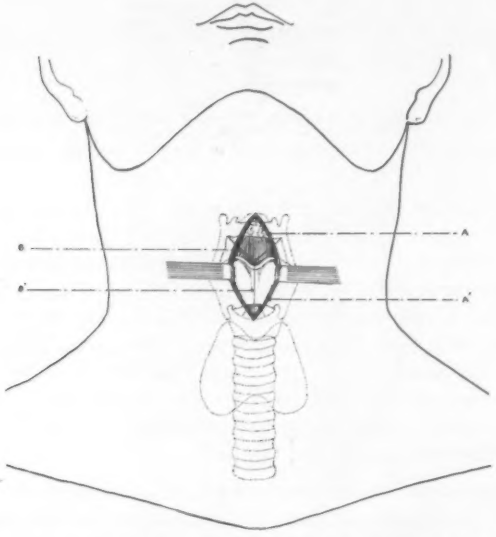

Fig. 8. Posterior View of Cartilages and Ligaments of Larynx (Cunningham's Anatomy).

muscles which are responsible for the various movements of which the larynx is capable.

Muscles: These muscles may be divided into the extrinsic and intrinsic groups.

The extrinsic muscles are responsible for the movements of the larynx as a whole, up or down. Those directly concerned in the upward movement are the thyrohyoid, inferior constrictor and stylopharyngeus muscles.

The downward movements are caused by action of the sternothyroid muscles and longitudinal fibres of the esophagus.[3]

Intrinsic Muscles of Larynx: It seems proper to include under this caption the cricothyroid muscles.

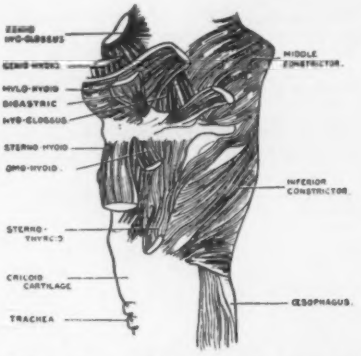

Fig. 9. Drawing Made From a Dissection of a Man, Showing the Extrinsic Muscles Acting on the Larynx (Negus, "Mechanism of the Larynx.").

The other important muscles will be considered from the point of view of action and will be so grouped.

There are two such groups, all of which pull on the arytenoid cartilages, the end result of their action depending on the points of attachment to the arytenoids.

A—Sphincteric Group: In analyzing this group it is found that it can be reduced to simple terms and so described as to be easily remembered. As stated before, all these muscles pull on or are inserted into the arytenoids. The cartilages of origin are the cricoid, arytenoids and thyroid. Hence the letters C A T may be used to designate the cricoid, arytenoid and thyroid cartilages, respectively.

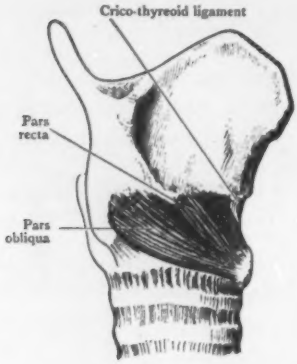

Fig. 10. The Cricothyroid Muscle (Cunningham's Anatomy).

This muscle has two parts: 1. Pars Recta. 2. Pars Obliqua.

Origin: Outer surface and upper lower border of cricoid ring near midline anteriorly.

Insertion: 1. Pars Recta — attached to lamina of thyroid cartilage (inner aspect of lower margin). 2. Pars Obliqua—anterior border of inferior cornu of thyroid cartilage.

Action: During respiration the anteroposterior diameter of glottis is increased. During phonation contracts in varying degrees, drawing thyroid cartilage forward and away from the cricoid and arytenoids. As a result of this the cords become tense.

Nerve Supply: External branch of superior laryngeal nerve.

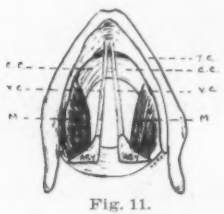

Fig. 11.

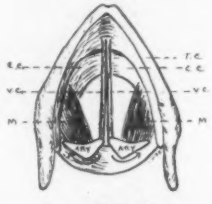

Fig. 12.

Fig. 11. The Cricoarytenoidei Lateralis Muscles (Relaxed).

T.C.—Thyroid cartilage. C.C.—Cricoid cartilage.

Fig. 12. The Cricoarytenoidei Laterales Muscles (Contracted).

V.C.—Vocal cords. M.—Cricoarytenoidei lateralis muscles. Ary.—Arytenoid cartilages.

Each muscle is triangular in shape and smaller than the cricoarytenoideus posterior.

Origin: Upper border of arch of cricoid cartilage extending to facet on lamina which supports the base of the arytenoid cartilage. Some fibres arise from the conus elasticus.

Insertion: Into the anterior surface of the muscular process of the arytenoid.

Action: When it contracts it (1) pulls arytenoid forward; (2) rotates arytenoid, causing approximation of vocal processes (adduction of cords).

Nerve Supply: Recurrent laryngeal nerve.

Using the arytenoid as a common point of attachment we therefore have the following combinations:

Cricoarytenoid (lateral).

Arytenoideus (inter).

Thyroarytenoid, or reduced to an anatomical formula.

Crico (lateral).

> Arytenoideus.

Thyro.

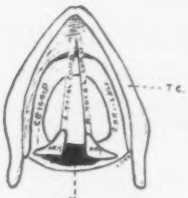

Fig. 13.

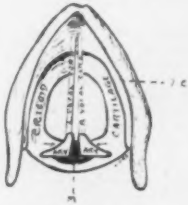

Fig. 14.

Fig. 13. The Arytenoideus Muscle (Relaxed).
T.C.—Thyroid cartilage.

Fig. 14. The Arytenoideus Muscle (Contracted).
M.—Arytenoidei muscles. Ary.—Arytenoid cartilages.

This muscle lies in the position of the posterior commissure.

1. Oblique Portion—Origin: Two bundles of fibres, each springing from the posterior aspect of the muscular process of corresponding arytenoid. They proceed upward and medially, crossing each other in midline.

Insertion: Some of the fibres are inserted into the summit of the arytenoid cartilage of the opposite side but most of the fibres are prolonged into the aryepiglottic folds and onward to the margins of the epiglottis.

2. Transverse Portion: Is an unpaired muscle, bridges the interval between the arytenoid cartilages, is attached to the posterior aspect of the lateral border of each arytenoid cartilage; many fibres turn around the arytenoids and become continuous with the fibres of the thyroarytenoid muscles (see Fig. 20).

Action: Slides the arytenoids towards each other, thus helping to close glottis posteriorly; also has function of closure during deglutition (together with rest of sphincteric group).

Nerve Supply: Recurrent laryngeal nerves (and internal laryngeal nerve).

For an understanding of these muscles you are referred to Figs. 10, 11, 12, 13, 14, 15, 16, 17 and 20.

Dilator Group: This is composed of a simple pair of muscles, viz., C A P, or *c*rico-*a*rytenoid *p*osterior. This pair abducts the cords (see Figs. 18, 19 and 20).

Vocal Cords: The description as given by Negus is worthy of repetition. In substance, he states that each cord is triangular in shape, pearly white in color and consists of yellow elastic tissue with

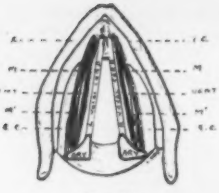

Fig. 15.

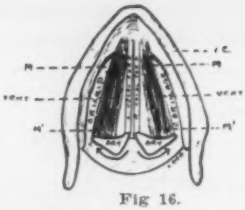

Fig 16.

Fig. 15. The Thyroarytenoidei Muscles (Relaxed).

T.C.—Thyroid cartilage. M.—Thyroarytenoideus muscle (inner portion). M'—Thyroarytenoideus muscle (external portion).

Fig. 16. The Thyroarytenoidei Muscles (Contracted).

Vent.—Ventricle of larynx. C.C.—Cricoid cartilage. Ary.—Arytenoid cartilages.

Origin: Lower part of inner surface of the thyroid cartilage near the midline and partly from the cricothyroid membrane; divides into two divisions: viz., 1. internal; 2. external.

Insertion: 1. Internal division into vocal process (tip and outer aspect). 2. External division into muscular process and concave external surface of body. Between these two divisions is a recess or laryngeal ventricle.

Action: 1. Draw arytenoids forward, relaxing cords when arytenoids are not braced posteriorly. 2. Make margins of glottis rigid when arytenoids are braced posteriorly, increasing elasticity of cord. 3. Adduct cords by rotating arytenoids, thus bringing vocal processes nearer to midline. 4. Almost complete apposition of ventricular bands by strong contractions of the external division.

Nerve Supply: Recurrent laryngeal nerve.

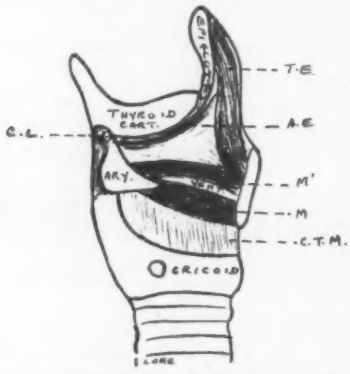

Fig. 17. Lateral View of Thyroarytenoideus Muscle (Left), Right Lamina of Thyroid Cartilage Removed.

T.E. — Thyro-epiglottideus muscle. A.E. — Arytenoepiglottideus ligament and muscle. C.L.—Cornicula laryngis. Art.—Arytenoid cartilage. M'—External portion of thyroarytenoideus muscle. M.—Internal portion of thyroarytenoideus muscle. Vent. — Ventricle of larynx. C.T.M. — Cricothyroid membrane (conus elasticus).

many fat cells in the meshes. It is extensible and acts as a covering to the thyroarytenoid muscle and provides a smooth surface capable of coming into firm contact with the cord of the opposite side. The

superior surface is flat and forms the floor of the ventricle. Some of the fibres of the thyroarytenoid muscle are closely bound to it and a few arising from the arytenoid cartilage are inserted into it.

These fibres constitute the aryvocalis muscle.

The cord itself extends from the internal face of the thyroid cartilage near the midline to the vocal process and part of the body of the arytenoid and is continuous below with the conus elasticus. It is covered medially by squamous celled epithelium (see Figs. 1, 4, 7, 21, 22, 31).

Laryngeal Ventricle: This space, which is horizontally placed, lies between the vocal cord and ventricular band, the upper surface of

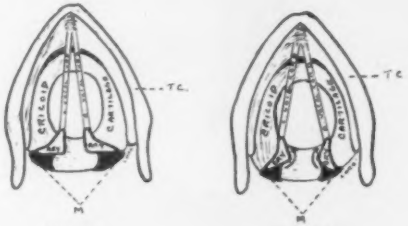

Fig. 18. Fig. 19.

Fig. 18. The Cricoarytenoidei Posterior Muscles (Partly Relaxed).

Fig. 19. The Cricoarytenoidei Posterior Muscles (Contracted).

T.C. — Thyroid cartilage. Ary. — Arytenoid cartilages. M. — Cricoarytenoidei posterior muscles.

These muscles are known as the abductors of the vocal cords; each muscle is fan-shaped.

Origin: Posterior surface of cricoid plate, fibres proceed upward and outward.

Insertion: Posterior aspect of muscular process of arytenoid cartilages.

Action: Rotate arytenoids so that vocal processes are brought away from the midline, thus opening the glottis, also cause arytenoids to slide outward.

Nerve Supply: Recurrent laryngeal nerve.

the cord and its extension externally forming the floor. This space also lies between the two divisions of the thyroarytenoid muscle. Anteriorly the roof of this space is extended upwards vertically to form a pouch, vis: the sacculus ventriculi laryngis. At this higher level the sacculus is located between the ventricular band and the inner surface of the ala of the thyroid cartilage (see Figs. 15, 16, 17, 21, 22 and 30).

Mucous Membrane: Lining the interior of the larynx we have mucous membrane. It can be stated as a general fact that the interior of the larynx is covered by columnar ciliated epithelium except those

parts which come in contact. These contact points are covered by squamous celled epithelium and may be enumerated as the vocal cords, margins of the ventricular bands (some cases), upper part

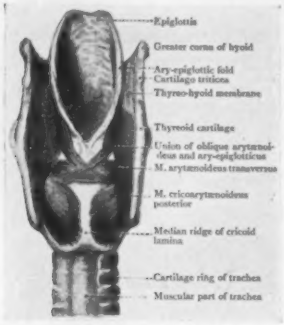

Fig. 20. Muscles of the Posterior Aspect of the Larynx (Cunningham's Anatomy).

See legends of Figs. 13, 14 and 18.

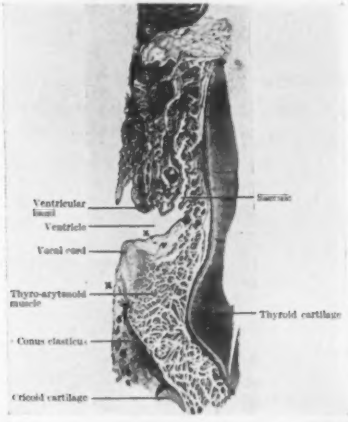

Fig. 21. Longitudinal Section of Larynx of a Child, to Show the Triangular Shape of the Vocal Cord and Its Relation to the Underlying Muscle.

The ventricular band, ventricle and saccule are also seen, together with numerous mucous glands. The mucous membrane between the crosses is squamous-celled (Negus).

of the internal surface of the epiglottis and aryepiglottic folds and the pharyngeal surface of epiglottis and rest of larynx[4] (see Figs. 21 and 22).

Nerve Supply—Motor: All of the intrinsic muscles are supplied by the recurrent laryngeal nerve with the following exceptions:

1. The interarytenoid muscle receives, in addition to the recurrent laryngeal nerve, some fibres from the internal branch of the superior laryngeal nerve.[3]

2. The cricothyroid muscle is supplied by the external branch of the superior laryngeal nerve.

Sensory: The internal branch of the superior laryngeal nerve supplies the larynx above the level of the cords while below this level the recurrent laryngeal nerve supplies sensory fibres. These

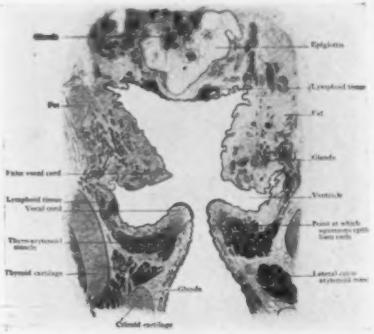

Fig. 22. Frontal Section of Larynx, About Middle of Vocal Cords (Piersol's Anatomy).
Note relationship of muscle to the vocal cord.

nerves communicate beneath the thyroid alae. These sensory nerves carry impulses to the brain where the motor fibres of the larynx take origin.

As a result of this stimulation, impulses travel down the different nerves to produce contracture of the laryngeal muscles, so bringing into play the protective mechanism (see Figs. 23, 24 and 25).

Blood Supply: Briefly stated, the interior of the larynx derives its supply mainly from: *1.* The superior laryngeal branch of the superior thyroid artery; *2.* The inferior laryngeal branch of the inferior thyroid artery. While the external branches of supply are from: *1.* The infrahyoid branch of the superior thyroid artery;

2. The cricothyroid branch of the superior thyroid artery (see Figs. 26 and 27).

Lymphatics: The description as given by Thomson and Colledge in their book on "Cancer of the Larynx" is so excellent that it will be quoted in part.

"1. The cavity of the larynx is divided into two lymphatic districts, an upper and a lower, which are almost completely separated from one another except by means of the lymphatics beneath the mucous membrane of the posterior wall. The dividing line of these two areas is the vocal cords, over which the lymphatics are very fine and few in number.

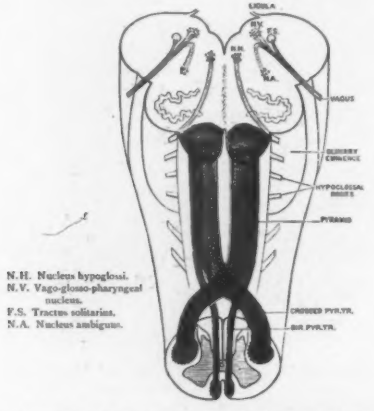

Fig. 23. Point of Exit of Vagus Nerve (Cunningham's Anatomy).
The central mechanism is located in the floor of the fourth ventricle and the upper border of the calamus scriptorius. Fibres originating here probably decussate before emerging from the brain stem.

"2. The lymphatics of the upper district collect in branches upon the side of the epiglottis, near the aryepiglottic folds, and leave the larynx through the pharyngoepiglottic folds and through the thyrohyoid membrane. The lymphatics of the lower district, which are of smaller calibre than those of the upper district, pass through the cricothyroid membrane. The lymphatics of this district communicate freely with those of the trachea. The lymphatics upon the posterior wall of the larynx, which communicate with both districts, communicate freely with the lymphatics of the esophagus and of the pharynx. The lymphatics in both areas communicate freely across

the median line, so that no barrier exists to glandular enlargement upon the opposite side of the median line."

When viewed in cross sections, the larynx gives information of considerable import. The method used by me has been first a study of the cross sections of the larynx at different levels and then taking X-rays of these sections and comparing the two. By means of the X-ray plates we can visualize what lies under the surface of the cross section.

Let us take a cross section of the neck, the thickness extending from just below the cricoid cartilage to the upper part of the thyroid cartilage. The first thing that strikes the eyes is that the larynx is virtually a rigid box, even though it is not a complete box at all levels.

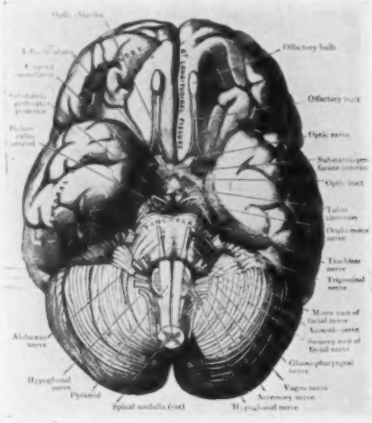

Fig. 24. The Base of the Brain with the Cerebral Nerves Attached (Cunningham's Anatomy).
Note the position of the vagus nerves.

The alae of the thyroid cartilage are worthy of note. They are comparable to a breast plate or armor plate. These alae extend posteriorly to a point well beyond the arytenoids so that all the structures of the larynx are well within their protective armor.

Lying between these alae and the aryepiglottic folds can be seen the pyriform fossae.

At a lower level the posterior wall of this rigid box is formed by the cricoid cartilage.

The practical importance of this arrangement becomes at once apparent, for any infection situated in the pyriform fossa must of necessity spread either posteriorly or medially.

The relationship of the carotid sheath and its contents to this rigid framework is also of importance. Experimentally it was found that infections starting at the posterior end of an ala of the thyroid were prone to reach the neck, after spilling beyond the limits of the larynx by extending laterally anterior to the carotid sheath, whereas infections starting in the lateral or posterior pharyngeal wall would travel posterior to this sheath (see Fig. 32).

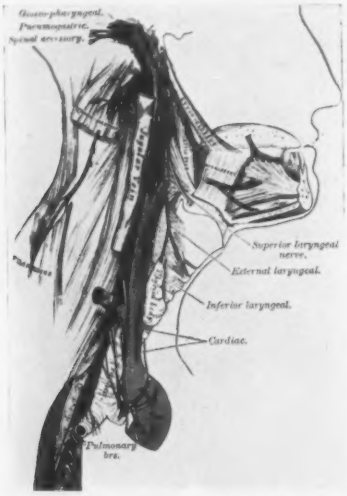

Fig. 25. Course and Distribution of the Vagus Nerve (Gray's Anatomy). In this illustration the superior laryngeal branch as indicated is really the internal branch of this nerve. Note that the right recurrent laryngeal nerve hooks around the right subclavian artery, whereas the left recurrent nerve hooks around the arch of the aorta.

Practical Considerations: Having reconstructed the larynx and studied not only its component parts, but also its general aspects, how can we apply this knowledge in a practical way? Anatomical landmarks are always of great importance and those furnished by the larynx serve a very useful purpose.

The upper border of the thyroid cartilage is approximately on a level with the fourth cervical vertebra and at this level the common carotid artery divides into its external and internal branches.

The mid-point of the anterior border of the thyroid cartilage denotes the level of the vocal cords.

The cricoid cartilage is at the level of the sixth cervical vertebra, at which level the esophagus begins. At this same level we have the carotid tubercle, which can be utilized for compression of the carotid artery.

The point of entry of the internal branch of the superior laryngeal nerve and the internal laryngeal artery through the thyrohyoid membrane can be determined by drawing a line across the upper level of the thyroid cartilage to a point within 1 cm. of the superior cornu

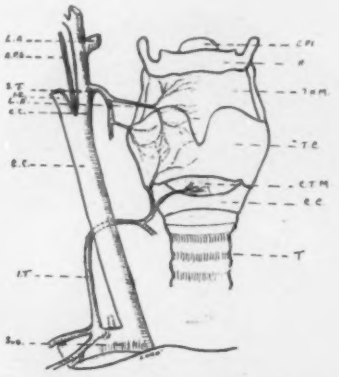

Fig. 26. Blood Supply of the Larynx.

L.A.—Lingual artery. A.P.A.—Ascending pharyngeal artery. S.P.—Superior thyroid artery. I.C.—Internal carotid artery. L.A.'—Laryngeal artery. E.C.—External carotid artery. C.C.—Common carotid artery. I.T.—Inferior thyroid artery. Sub.—Subclavian artery. Epi.—Epiglottis. H.—Hyoid bone. T.H.M. — Thyrohyoid membrane. T.C. — Thyroid cartilage. C.T.M. — Cricothyroid membrane. C. C.—Cricoid cartilage. T.—Trachea.

Note that the superior thyroid artery is a branch of the external carotid artery, while the inferior thyroid artery is a branch of the subclavian artery.

of this cartilage. These are the most important landmarks and have great practical importance (see Fig. 33).

Now let us turn our attention to some of the more important practical significances of the anatomy of the larynx.

Cut Throats: These wounds are usually self-inflicted and as a rule involve the thyrohyoid membrane. A wound transversely across this membrane amounts to a subhyoid pharyngotomy. Through this wound can be seen the posterior pharyngeal wall, one or both pyriform fossae and the upper introitus of the larynx. If the wound

is just above the level of the thyroid cartilage and has been carried sufficiently lateral, it may have involved the sensory nerve of the larynx and its accompanying artery. In such a case the protective reflex mechanism of the larynx (sensation) will have been impaired and the bleeding may be so severe that the patient may drown in his own blood. In such a case immediate control of the bleeding, establishment of an open air-way and the introduction of a feeding tube are essential.

Immediate management of such a case will depend on circumstances. In an emergency, where the air-way is impaired and minutes mean much, it is justifiable to introduce a long tracheotomy

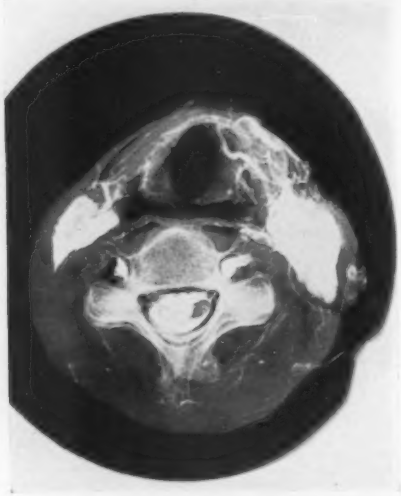

Fig. 27. Injected Cross-section of Neck, Including Cricoid Cartilage and Hyoid Bone, Showing Blood Supply of Larynx.

tube through the neck wound between the vocal cords. This will provide sufficient air-way and at the same time prevent blood and secretions from being aspirated. All bleeding having been controlled, a leisurely tracheotomy may then be performed. In one of the three cases which I have had in which the tracheotomy tube had been introduced between the cords, no tracheotomy was found necessary. If no edema of the larynx is present, a tracheotomy will in all probabilities not be required.

Incisions in Thyrohyoid Membrane: A knowledge of the anatomy of this region simplifies the placing of incisions to approach the hypo-

pharynx, epiglottis or the larynx. This knowledge has been utilized in modifying the laryngofissure operation by extending the incision through the thyroid cartilage into the thyrohyoid membrane by means of a V-shaped incision. The important points to remember in this region are: *1.* the position of the epiglottis, and *2.* the point of entry through this membrane of the internal branch of the superior laryngeal nerve and the internal laryngeal artery (a branch of the superior thyroid artery).

Incidentally, it may be stated that with the aforementioned information it becomes a simple matter to inject or infiltrate any solution about this nerve for purposes of anesthesia or therapy.

Injuries to Larynx: The larynx proper is supported and protected by the thyroid cartilage, a strong cartilaginous shell. Fractures or

Fig. 28. Lymphatic Glands of the Neck.
(Prof. Dr. M. Hajek, Vienna).

infections of this rigid framework may and often are attended by serious consequences, the most important of these being encroachment on the air-way producing dyspnoea and even asphyxia. Knowing the mechanism of the larynx, the management of these cases becomes clear. The first requisite is to establish a free air-way and this is done by means of a tracheotomy. This done one may then direct his attention to a reduction of any fractures, hematoma or infection resulting therefrom. The question of laryngeal infections will be considered separately.

The cricoid cartilage, which is a strong cartilaginous ring, should be considered as the base on which the larynx rests and any injury

or infection involving it may lead to a stenosed trachea or larynx. It is important to bear in mind in performing a tracheotomy that this structure is not injured.

Infections of the Larynx: The relationship of the soft structures to their rigid framework influences the spread of infection and the ensuing symptoms. Any infection in the larynx is characterized by marked and extensive edema, all out of proportion to the amount of infection. Due to the cartilaginous barrier, infections cannot spread equally in all directions, but will follow the path of least resistance. Clinical observations and experimental work on the cadaver show this to be true. Solutions injected into the laryngeal tissue, anterior

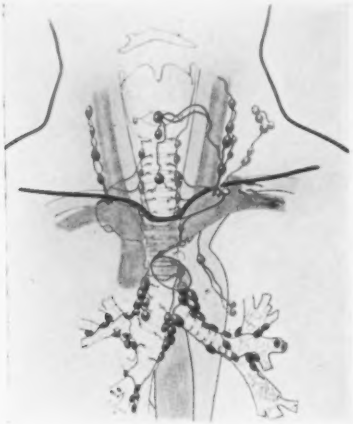

Fig. 29. Lymphatic Glands of the Larynx, Trachea and Bronchi (Prof. Dr. M. Hajek, Vienna).

to the arytenoids, will spread inside the larynx, whereas those injected more posteriorly or posteriorly to the posterior edge of the thyroid cartilage may extravasate laterally into the neck, either anteriorly or posteriorly to the carotid sheath. Therefore, infections in the anterior part of the larynx do not give external manifestations as early or as marked as in infections situated more posteriorly. Infections medial to the aryepiglottic fold are more apt to extend medially, producing severe edema and laryngeal obstruction, whereas infections external to this aryepiglottic fold are apt to spill into the pyriform fossa, causing its obliteration. The dangers of laryngeal obstruction, while present and great, do not approach those produced by

infections more medially located. Pain, however, is apt to be more marked.

Treatment depends on the severity of the symptoms. Laryngeal edema with marked obstruction to the air-way often demands tracheotomy. With a clear air-way re-established, treatment of the local condition becomes easier. If an abscess is suspected, incision and drainage is indicated, using the direct laryngoscopy method.

In less severe cases operation is not indicated and the condition may respond to local measures.

Where the infection has spilled outside the larynx and extended into the neck, external approach is indicated.

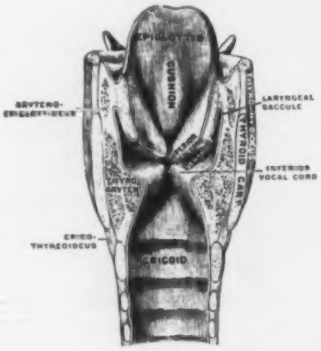

Fig. 30. Coronal Section of Larynx, Rear View of Front Half (Testut). Note position of ventricle and laryngeal saccule.

Paralysis of Vocal Cords: Any involvement of the nerves supplying the larynx may produce some paralysis. By referring back to this nerve supply (see Figs. 23, 24 and 25), it will be noticed that the nerves are vulnerable to disease or trauma at many points. Constitutional diseases as syphilis or tuberculosis may cause paralysis. Enlarged lymphatic glands, thyroid gland (benign or malignant), apical tuberculosis involving the pleura, especially on the right side (causing adhesions between the pleura and right recurrent laryngeal nerve), carcinoma of the esophagus or mediastinum and dilated arch of the aorta or aneurism are not infrequent factors. A study of these lesions is a very imposing one and of a scope too extensive to permit a detailed consideration for this presentation. Therefore, a rather sketchy description will be given.

Central: The more common conditions affecting the vagus centrally are softening, hemorrhage, tabes, bulbar paralysis and multiple sclerosis. The effect on the larynx is complete paralysis with loss of sensory function. Central involvement of this nerve often includes involvement of the ninth, eleventh and twelfth, causing paralysis of the soft palate and impairment in swallowing.

Peripheral: (a) Involvement of Superior Laryngeal Nerve: If involved by direct or indirect causes in its entirety before giving off the internal and external rami, the disturbances would be both sensory and motor.

1. Sensory: The protective mechanism of the larynx would be impaired so that food or secretions would have a tendency to collect in the pyriform fossae and enter the larynx.

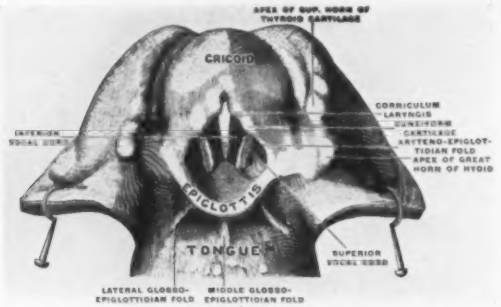

Fig. 31. Larynx Viewed from Above (Testut).

2. Motor: The result here would be essentially a loss of tension of the vocal cords and the inability to control the voice because of the inaction of the cricothyroid muscles and possibly a triangular gap between the vocal processes anteriorly and the posterior commissure posteriorly. However, the fact that the interarytenoideus muscle also receives innervations from the recurrent laryngeal would make this unlikely or at best incomplete.

In addition there may be immobility of the epiglottis.

Paralysis of this nerve (superior laryngeal) may be caused by new growths, enlarged glands or wounds.

(b) Involvement of the Inferior (Recurrent) Laryngeal Nerve: The end-result is an abductor paralysis. In cases where this nerve is gradually destroyed, as by pressure from a new growth, the

paralysis of the muscles follows a definite order (Semon's Law). Abduction is first affected through disturbed action of the posterior crico-arytenoids. The thyroarytenoids are next involved, and then the lateral cricoarytenoids. The arytenoid, unimpaired, has a bilateral nerve supply and is therefore not affected in unilateral cases.[6]

Adductor Paralysis: This merits separate mention. According to Osler this form of paralysis leading to difficulty or abolition of phonation forms a distinct class of functional paralysis. "It may be said that adductor paralysis is never due to organic disease in the passage of the nerve from the bulbar muscles to the larynx, but is either a pure neurosis or due to local disease." "This form of paralysis is almost always bilateral and seldom complete. There are three sets

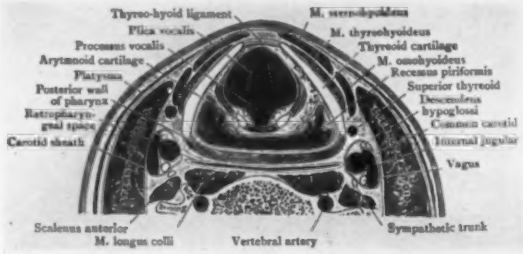

Fig. 32. Transverse Section Through the Neck at the Level of Upper Part of Thyroid Cartilage (Cunningham's Anatomy).

Note particularly: 1. Protection of larynx by laminae of thyroid cartilage. 2. Pyriform fossae in relation to laminae of thyroid cartilage. 3. Position of carotid sheath and contents.

of adductor muscles, the cricoarytenoids (lateral), the arytenoideus and the thyroarytenoids."[7]

Surgery of the Vocal Cords: An understanding of the vocal cords brings one to the realization of wonderful surgical possibilities. Predicated upon this anatomical concept and clinical observations made in cases where the cords have been removed in toto or in part, some cadaver, animal and human studies have been made by me in "avulsion of the vocal cords."

Deliberate stripping of the vocal cord in indicated conditions has resulted in such remarkably good results that further application of the procedure is being studied and will be the subject of a later presentation.

Suffice it to say that the mucous membrane, or the fibrous elastic substructure or both will regenerate after removal.

Blood Supply: The practical application of the knowledge of the laryngeal blood supply is demonstrated in performing a laryngectomy. Knowing where the main vessels of supply are located, troublesome bleeding can be avoided by proper ligation. In the laryngofissure operation there is a fair sized vessel which is often encountered.

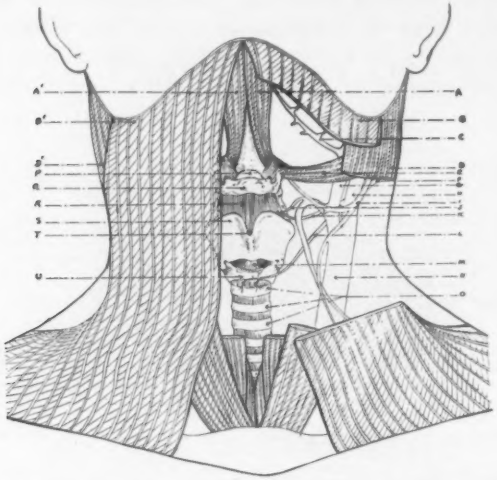

Fig. 33. Semidiagrammatic Drawing of Anatomy of Neck (Front View). A-A.'—Anterior bellies of digastric muscles. B-B.'—Platysma muscles. C.—External maxillary artery. D-D.'—Sternomastoid muscles. E.—Posterior belly of digastric muscle. F.—Superior laryngeal nerve. G.—External carotid artery. H.—Internal carotid artery. I.—Hypoglossal nerve. J. — Internal laryngeal branch of superior thyroid artery. K. — Internal branch of superior laryngeal nerve. L.—External branch of superior laryngeal nerve. M.—Cricothyroid membrane. N.—Common carotid artery. O.—Tracheal rings. P.—Tip of Epiglottis. Q.—Hyoid bone. R.—Thyrohyoid membrane. S. — Thyroid cartilage. T. — Thyroid notch. U. — Cricoid cartilage.

This is located anterior and external to the vocal process of the arytenoid on the operated side. While pressure will usually control the bleeding from this vessel, secondary hemorrhage is not infrequent. Hence the importance of realizing its existence and ligation. In using the Loré technique in this operation, clamping and ligation of this artery become a simple procedure.

Lymphatics: The vocal cords, anterior to the vocal processes, are almost devoid of lymphatics. Therefore, any growth situated here

will be slow to metastastize. As a lesion approaches the posterior commissure or spreads either above or below the cords, the dangers of metastasis increase and it is important to bear this in mind in prognosticating the end results in such conditions.

Fortunately, carcinoma of the larynx is more apt to be situated anteriorly, whereas tuberculosis of the larynx is as a rule more posteriorly.

Another fact of considerable practical importance is that while the original lesion in the larynx is on one side, metastasis to the

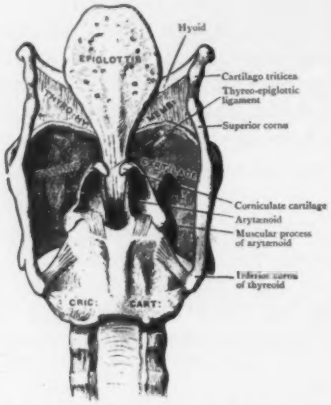

Fig. 34.

A-A.'—Skin incision for laryngofissure operation without preliminary tracheotomy. B-B.'—Incision in thyroid cartilage and thyrohyoid membrane.

glands may be on the opposite side. This is particularly true if the original lesion involves the arytenoids or posterior commissure.

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325 West End Avenue.

SYMPOSIUM ON THE LARYNX.

II.—ETIOLOGY AND TREATMENT OF CONTACT ULCER OF THE LARYNX.*

DR. CHEVALIER LAWRENCE JACKSON, Philadelphia.

Contact ulcer was first described by Chevalier Jackson in 1928, at which time he stated that he had seen 217 cases in forty years. He defined the lesion as "a superficial ulceration occurring on one or both sides of the larynx posteriorly, the ulcerated surface coming in contact on phonation with that of its fellow of the opposite side, the latter being ulcerated or not, according to whether the ulcer is monolateral or bilateral." The term "contact ulcer" is reserved in its strictest use for benign nontuberculous lesions, but such lesions may become cancerous, and similar lesions may occur in a frankly tuberculous laryngitis.

Our conception of the pathology of contact ulcer has heretofore been based on clinical examination and biopsy. Recently, however, a very important contribution to the study of this condition has been made by Dr. Achille Peroni, of Milan, Italy. In a paper about to be published, Dr. Peroni reports two cases of contact ulcer studied at autopsy in the Instituto di Anatomia Patologica of the University of Milan.

The present paper is based on a careful study of the records of ten typical cases seen within the past three years.

Etiology: Occupation was the first matter considered and in several of the cases it was probably a factor, in that the occupation required excessive use of the voice. One patient was a salesman, two were physicians who did a considerable amount of teaching and lecturing, another held a clerical position which required him to interview several hundred people every day. Tobacco and alcohol have been regarded as important etiologic factors, and yet I found that only about half of the patients in this group admitted their use. Two patients were heavy smokers, two moderate, and one only an occasional smoker. Four admitted the occasional use of alcoholic

*Read by invitation before the New York Academy of Medicine, Section on Otolaryngology, Feb. 15, 1933.

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beverages and one said he had "been quite a heavy drinker until about twenty years ago." Focal infection in the throat and upper air passages was apparently a contributory factor in almost all the cases. Eight of the patients had either a postnasal discharge or infected tonsils. Two had had a Vincent's infection. Five had cough, which might be regarded as a symptom, but was probably a pre-existent etiologic factor in most cases. In six cases the onset of laryngeal symptoms dated from an acute respiratory infection. One of these six patients attributed his trouble to shouting while deer hunting during an acute respiratory infection. In one rather unusual case the lesion developed after paralysis of the left cord subsequent to an operation for aural vertigo. The ulcer developed on the opposite cord, at the site of impingement of the vocal process of the paralyzed side. Sex constitutes an etiologic factor that should not be ignored; all these ten patients were men, though the condition is also observed in women. Summarizing, then: occupation, vocal abuse, tobacco, alcohol and infection would seem to be among the important etiologic factors. It might be added that most of the patients had a rather "throaty" method of voice production, and that their average age was about fifty years.

Treatment: Unquestionably the most important item in treatment is vocal rest. This will suffice in many cases to effect cure, but it should usually be continued for some time after apparent cure, because to obtain permanent healing of any lesion where there has been necrosis of cartilage requires a long time, and in advanced contact ulcer there is almost always necrosis of the tip of the vocal process of the arytenoid. The ulcer will never heal until the cartilage has healed and been covered over. Six or eight months is not long for cartilage to heal.

Vocal rest may be supplemented by the instillation of some bland oily medicament if desired, but we feel that all irritant substances or caustics should be avoided. To quote from Chevalier Jackson, "Energetic ill-advised treatment is usually followed by edema, loss of cartilage, impairment of the cricoarytenoid joint and increased hoarseness. In one patient sent to us, extensive cauterization had caused perichondritis, with acute edematous stenosis followed by chronic cicatricial stenosis." Sea air has seemed to be beneficial in some of the cases; one of the patients in the series is spending the winter in Florida with benefit. In a certain number of cases excision is advisable, as previously mentioned, both for diagnosis and for treatment. The hyperplastic tissue about the ulcer is curetted

off with delicate laryngeal forceps used through the direct laryngoscope, and the tissue should always be sent to the laboratory for histologic study. Excision was done in four of the ten cases analyzed. In excision, care should be taken not to damage the cricoarytenoid joint, but there is no objection to taking off a bit of the tip of a necrotic vocal process.

Finally, etiologic factors such as tobacco, alcohol, dust, fumes, as well as foci of infection in the sinuses, faucial or lingual tonsils, or teeth, should of course be eliminated or minimized.

As for results obtained, it may be stated that most patients get entirely well, but they usually require a long period of treatment and are prone to develop recurrences when they again subject the larynx to abuse, especially if they do not continue the regime of vocal rest quite long enough. The ten cases studied are too recent for final statistics to be reported, but in two instances the lesions were completely healed when last seen, and most of the others have showed progressive improvement.

Replying to Dr. Kernan in re: ulcers of vocal *cord*, I quite agree that we should not speak of contact ulcer as an ulceration of the *cord*, because it is chiefly the cartilaginous glottis that is involved.

Answering Dr. Settel in re pachydermia, the location of contact ulcer is very much the same as that of pachydermia, but these patients have nearly all been seen early, when there was just a little ulceration; the building up of tissue about the ulcer was the second phase, so that I do not feel that the cases of contact ulcer may be regarded as ulcerated pachydermias.

Dr. Lloyd's idea of keeping the cords apart by means of some sort of apparatus seems a very good one; to do this would be carrying the idea of vocal rest one step further.

Replying to Dr. Scheer, in many of the cases there is a building up of hypertrophic tissue around the ulceration, and in a number of them this constitutes a sort of granuloma, probably originating from the cartilage or perichondrium. We see similar granulomata in other conditions where there is an infection of cartilage. In contact ulcer we may see an ulcer on one side and a granuloma on the other side fitting into it. The granuloma may disappear from one side and a similar one appear on the other, etc. It is frequently necessary to take these granulomata off instrumentally at direct laryngoscopy. This was done in four of the ten cases. In one or two others, it could easily have been considered good treatment to do likewise.

The suggestion of Dr. Mandelbaum seems a very plausible one and it may be the explanation of the etiology in some cases. It is likely that the mechanism is not the same in all cases. In one of the cases in our series there was a paralysis of the left cord, with a jutting inward of the vocal process in such a way that a contact ulcer was produced on the opposite side by the constant irritation.

I agree with Dr. Myerson that we see a majority of laryngeal lesions due to vocal abuse on the middle third of the cord. Just as in other conditions, however, one patient may have one weak point while another has another weak point; in most patients this may be in the middle third of the cord, while in the patients who develop contact ulcer it is in the cartilaginous glottis. These latter patients must have a *locus minoris resistentiae* in this region, and the reason that they develop contact ulcer rather than some other lesion probably lies in the anatomy and physiology of the part.

3432 North Broad Street.

SYMPOSIUM ON THE LARYNX.

III.—DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE LARYNX.*

DR. JOHN D. KERNAN, New York.

One easy way to write an essay on the diagnosis of diseases of the larynx would have been to take a textbook, copy its conventional list of diseases of the larynx and the points to be considered in their differential diagnosis, jumble it all up so that it would not look too familiar and then read it to you. I thought, however, that in all probability most of you are more familiar with the textbooks than I am and that such a procedure, though doubtless profitable to me, would help you not at all, even as a basis for discussion. It seemed to me, therefore, that it would be better to discuss diseases of the larynx comparatively little, and to talk rather of the methods of diagnosis and the extent to which each is helpful or, on the other hand, misleading.

Considered from this point of view, I have found that the more help one has, the harder to make a diagnosis, until finally it is almost impossible.

Diagnosis of diseases of the larynx began with the invention of the laryngoscope. That seemed to solve all problems. What could be easier or simpler when one saw a patient with loss of voice, difficulty in breathing or pain, than to look into the larynx, see what was there, and so know what the trouble might be? In actual fact, however, it does not prove to be so simple. Think of the almost numberless conditions disclosed by the laryngeal mirror alone, the paralyses, inflammations, tumors, ulcers and neoplasms. Instead of having one problem on his hands, the physician has a dozen. Suppose the question has arisen as to why the patient is hoarse. The mirror shows a tumor. There is the answer. The patient is hoarse because he has a tumor of one of his vocal cords. But wait a minute! What kind of a tumor is it with which we are dealing? Is it benign, malignant or inflammatory? If benign, is it a simple fibroma that can be removed with a bite of the forceps, or is it a

*Read as part of a Symposium on the Larynx before the New York Academy of Medicine, Section on Otolaryngology, Feb. 15, 1933.

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papilloma of such tendency to recur that the whole base must be removed? Supposing it is malignant, is it locally so or will it rapidly metastasize? Is it of slow or rapid growth? Is it radio-sensitive? How do you know it is malignant? How do you know it is not syphilitic or tuberculous or a keratoma or a pachydermia? How, having seen the tumor, are you going to answer any one of the dozen questions which assail your poor brain? The answer must be sought as carefully as that to any other question in diagnosis. You must consider the age of the patient, take a careful history, bar out syphilis and tuberculosis (and by this I mean taking a daily record of temperature, physical examination, sputum examination and chest X-rays), and perhaps do a biopsy. Then when all these things are done, you may be wrong after all, as I shall show you later.

Now, the point I am trying to drive home to you is this: You must not expect to find your answer to a question of diagnosis of any disease of the larynx in the larynx alone. You will find it in the whole of the patient. In others words, you must be primarily a good physician and then you may hope to be a good laryngologist.

Suppose that, in your first examination, you handle the mirror perfectly, that you see the larynx well, and yet you can see no cause for the attacks of weak voice, the constant clearing of the throat, or the occasional hoarseness. Suppose that one cord lags a little and yet the mirror shows no cause for it. You must remember that there are regions in the larynx which the mirror will not show. One cannot see into the ventricles with the mirror. Perhaps the anterior commissure is hidden by a stiff epiglottis. The undersurface of the cord may be invisible. What is the answer? Direct laryngoscopy. You may say that this procedure is too formidable. You may say to yourself, "I cannot send my patient needing a direct laryngoscopy to a hospital." My answer to that is that comparatively few cases will need direct laryngoscopy. Those that do, need it badly and if the individual laryngologist is not prepared to make the examination himself, he should ask for help. I say this further. I do not believe in direct examinations in the office, when great refinement of technique is called for. For such a procedure, the head must be held just so, the anesthesia must be correct and the patient psychologically controlled. At times, even a general anesthesia may be called for. All these things require a technique and teamwork that only a properly equipped bronchoscopic clinic can give.

Do not understand from what I have said that I do not believe that every laryngologist should be prepared and able skilfully

to use the direct laryngoscope. I do. Every laryngologist should be able to examine a larynx with the direct laryngoscope, especially in acute conditions in children. You all know how difficult it is to see with the mirror in children. It is impossible in infants. Therefore, in acute conditions in young children, the use of the direct laryngoscope is absolutely necessary. How quick and sure is the answer then? Is the condition laryngeal diphtheria, catarrhal croup or streptococcus laryngitis? Is it a foreign body? Is a culture needed? These problems are solved at once.

The question which is put up to me most frequently nowadays is that of dyspnea in very young infants. I have seen a number of youngsters lately; one three weeks old, whose history was that of inspiratory crow since birth with retraction above the clavicle and below the ribs. What could it be? We might find a congenital web, a papilloma, a compression of the trachea by the thymus. The thing I see most frequently in such cases is just a collapsing orifice. Epiglottis, arytenoids and aryepiglottic folds all seem to suck in with inspiration. The children get over it with age. Another condition which is common is the congenitally narrow subcordal space. Tucker has described it and devised an instrument for measuring that part of the larynx. The condition may be so extreme as to call for a tracheotomy. I did one recently on a young infant who has gotten along very well. The question of tracheotomy arose in the baby three weeks old whom I mentioned, but as it was gaining weight, we held off and it has continued to improve. This about covers the ground as far as laryngeal difficulties in children are concerned.

Now, we must go to the troubles of adults. The larynx, as you know, serves two purposes: talking and breathing. Talking is the function most frequently affected; in other words, hoarseness is the most common symptom of laryngeal disease. The patients who come to a laryngologist because of shortness of breath have usually been hoarse for months.

A patient comes into your office complaining of loss of voice. A few questions bring out the fact that the hoarseness has been of considerable duration. In other words, you may rule out simple acute laryngitis. On examination, you may see some form of simple chronic inflammation. I need not describe to this audience the appearance of such a larynx, the cords thick and red and covered by thick mucopus, or perhaps dry and shiny. On vocalization, they move freely and meet well, but their vibration is not up to normal and hence the hoarseness. Is your diagnosis made? Not at all. What

is the underlying cause of this chronic laryngitis? Is it bad tonsils, bad teeth, bad sinuses, liquor or tobacco? Is it bawling or singing or any other straining of the voice? All of these questions must be asked and all habits investigated before the diagnosis can be made. It may not be a simple laryngitis after all, but rather some rarer form of chronic inflammation, such as hyperkeratoses or pachydermia laryngis. These latter conditions must be more or less rare as I have seen very few cases of either.

Perhaps the case may prove to be one of the specific chronic inflammations, such as syphilis, tuberculosis, rhinoscleroma or leprosy. The latter two are certainly rare in these parts but must be considered. The diagnosis would probably be made on lesions elsewhere. Syphilis and tuberculosis will be considered later.

A benign neoplasm of some kind may be found, such as a papilloma, angioma, fibroma, singer's nodules, etc. These, as a rule, are easily identified. They are likely to be more or less pedunculated, noninfiltrating, freely movable and do not interfere with cord movement. One removes them without hesitation and the microscope settles the issue.

Perhaps a paralysis or paresis is revealed by the mirror. I am sure that we all see many cases of long continued hoarseness in which the cords just do not fit. The vocal processes approximate promptly but the edges of the cords do not meet. Intrinsic weakness of the cords, as I call it. Hysteria is also a common cause of loss of voice. In women, many of these cases occur and they are characterized by a long history of sudden loss and return of the voice.

The real paralyses are different matters. A prolonged and painstaking study may be called for. It must be determined whether the causative factor be central or peripheral. If peripheral, one must account for the whole length of the recurrent nerve. Is there pressure from an enlarged thyroid gland, from cervical glands, from a tumor in the mediastinum, from an aneurism or from an enlarged heart? I have seen a left cord paralysis from an enlarged right auricle. The diagnosis was made in that case by bronchoscopy, the left main bronchus being compressed just where the recurrent nerve would round it. The auricle was the only structure in just the position to make the pressure. Recently I saw a paralysis of the right cord in a young girl. The only way in which we could account for it was to consider it due to a thickened pleura at the right apex. She had a tuberculous history.

Within the month, a middle-aged man came to the clinic, giving a history of dyspnea and severe choking attacks. Examination revealed a double abductor paralysis. The full diagnosis was not made until a serological examination proved him to be a victim of locomotor ataxia. I have seen other laryngeal paralyses which were due to encephalitis lethargica.

Chronic simple inflammations are amenable to treatment and subside. Benign tumors are easily removable. Paralyses can be borne without too great alarm. Very different is the situation when the question of cancer arises. There is an enemy which is grim indeed and the diagnosis must be sure and early. Let me repeat the *early*. Anyone can spot a late, hopeless cancer.

A man of middle age gives a history of hoarseness of some standing. What will lead one to think it cancer? There is a small tumor on one cord. Can a diagnosis be made on its appearance alone? About twenty years ago, Sir Felix Simon[1] reported three cases showing laryngeal lesions so closely resembling one another that they could not be differentiated by the eye alone. One was syphilitic, one tuberculous and one carcinomatous. Evidently observation alone will not always do. Cancer appears anteriorly on one cord, never on both simultaneously. It may take the form of a papilloma, a shallow ulcer or a localized thickening of the cord. It has a tendency to shade off into the surrounding tissue, unlike benign tumors. There is no inflammatory areola about it and there is no pain or dysphagia. Immobility of the cord is often mentioned as an early sign. According to St. Clair Thomson,[1] immobility of the affected part shows in less than half of the early cases. It is a late sign and means infiltration of the surrounding tissues. Moreover, immobility of the cord can also be caused by syphilis or tuberculosis.

Tuberculosis begins in the interarytenoid space and is bilateral and symmetrical. Pain and dysphagia occur early and fixation of the cords is very late. Also, tuberculosis is said never to be primary in the larynx. Careful search will reveal a lesion in the lung or bacilli in the sputum.

Syphilis is liable to be widespread in the larynx. It develops rapidly and ulceration is early. Then, too, we have the Wassermann reaction to help us, as well as the therapeutic test. A syphilitic lesion will dissolve rapidly under treatment.

Two other classes of carcinoma of the larynx must be considered. Tumors will occasionally develop in the ventricle, more frequently

on the under-side of the cords. The symptoms are obscure in these cases. Early hoarseness does not occur. There may be an infrequent huskiness of short duration. Some patients will complain of uneasiness in the throat or of voice fatigue. Examination with the mirror will show nothing until quite late as the growth is hidden. Only a most careful direct examination will reveal the tumor.

It all seems quite simple. Given a suspicious lesion in the larynx, if tuberculosis be found elsewhere, then the lesion is tuberculous. If the Wassermann reaction be positive, the lesion is syphilitic. In reality, it is not so simple. Tuberculosis and carcinoma may occur in the larynx together. So may syphilis and tuberculosis. So for that matter may all three. What must we do? St. Clair Thomson recommends long and repeated observation. Syphilis and tuberculosis develop more rapidly than carcinoma. The latter is unaffected by rest and treatment. If, after a proper interval, the lesion continues a slow development, it is most likely to be a carcinoma.

Why should we not always do an early biopsy in these cases? There is a decided prejudice against biopsy in carcinoma cases on the part of most surgeons. The late Dr. MacKenty was decidedly opposed to it. However, there is some experimental evidence which appears to show that the prejudice is not justified. In 1919, F. C. Wood[2] published the results of experiments on rats in which the rat carcinoma had been transplanted. These experiments showed that metastases were not increased when the tumors were incised, a fragment removed and the growth allowed to remain in the animal for from ten to twelve days thereafter. Wood, however, promptly kicks his child outdoors, stating that a biopsy should not be performed unless the diagnosis can be made in no other way and it should be followed promptly by operation if carcinoma be shown. Those interested in the present status of biopsy should read Hellwig's latest article.[3]

There is further reason for biopsy. These tumors must be graded. According to the grading will the prognosis vary. Gordon B. New,[4] reporting at the 1932 meeting of the American Medical Association, showed that the prognosis was much more favorable in the tumors which graded low in the scale of malignancy. In addition to this, there is the question of radiotherapy. The Roentgenotherapist will call for a biopsy as he bases his prognosis on the histology of the tumor. For these reasons, a biopsy seems justified, both from the standpoint of diagnosis and of prognosis. It must, however, be done with the greatest care, preferably by the direct method. A

poorly selected piece may miss the tumor entirely and a roughly taken piece may cause metastases.

Now, suppose that we have made our biopsy and have received a report of carcinoma, graded by a skillful pathologist. Are we permitted to enjoy our triumph? Not at all. In the first place, Stanley Reimann published an article[5] on the grading of tumors of the breast. He took one hundred cases of breast carcinomata, the final results of which were shown. According to him, it was impossible to tell from the histological appearance of the tumors which patients would live and which would die. For instance, he took sections from the seventeen living patients, mixed them with sections from seventeen patients who were dead, and found that he was 50 per cent wrong in his attempt to pick out the favorable cases. Patients react differently to tumors which appear exactly the same to the diagnostician.

Now as to the diagnosis of carcinoma on biopsy specimens. I have had some experience in connection with carcinoma of the bronchus. I have removed specimens from the bronchi of three children under 15 years of age. All three tumors were pronounced carcinomatous and yet all three have been nonmalignant clinically. Two have disappeared in response to diathermy. The other is still present in the lung at the end of twelve years and yet the patient is healthy and without symptoms. Evidently these patients have carcinoma but it is not the disease carcinoma.

Some of you may question the transference of these conclusions to the problem of carcinoma of the larynx. I wish to call your attention to a paper by Harrington B. Graham,[6] in which he reports one case of a boy from whose larynx a papillary carcinoma was removed by stripping off the cord. In a month's time, the larynx appeared almost normal and free of growth. This case was histologically malignant but clinically benign. In the same paper is reported the case of a man of seventy in whose larynx had been a proven carcinoma for twelve years. The man lived ten years longer. Obviously, that was not a clinically malignant growth. Graham reported a number of other cases of tumors, histologically malignant, in which local removal seemed to control the growth.

Where are we now? Having called for a biopsy as the final test in the diagnosis of laryngeal carcinoma, I have shown that this procedure is reliable neither for diagnosis nor for prognosis. Evidently much work lies ahead in the development of laboratory meth-

ods before they can be trusted to settle the question. Tests must be devised which will allow the differentiation of the clinically non-malignant from the malignant cases. Then we will know which require laryngectomy, which thyrotomy, which local removal and which Roentgenotherapy.

In the meantime, what must we do? We must take a careful history, observe and study carefully and patiently. We must examine every part of the larynx with mirror and direct laryngoscope. Then, if doubtful, prepare for operation and do a biopsy.

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DIATHERMY: SURGICAL AND MEDICAL IN OTOLARYNGOLOGY.*

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It has become evident that with the improvement in the high frequency apparatus, diathermy has a definite place in otolaryngology. The early machines produced some Faradic current, which caused violent contractions of the muscles, and sharp pain in the adjacent sensory nerves. The manufacturers of the better types of apparatus have largely overcome this drawback.

Though diathermy is in its comparative infancy, it is sufficiently well perfected to be practical. There are diathermy machines now available which will generate the three high frequency currents, namely, the cutting, the coagulating and the dessicating, in a satisfactory manner. While they are still far from absolutely perfect, one need not go deeply into the electrophysics of choke, primary and secondary coils, spark gaps, d'Arsonval coil, Oudin coil, condensers, high voltage, low amperage, etc. All you require is that the apparatus shall give the quality and necessary volume of current, and that you study the effects of the currents.

One is confronted with reams of pseudoscientific literature mainly in second class journals, by otherwise unknown authors whose statements are overenthusiastic, immoderately narrow and dogmatic. This is augmented by syndicated medical columns in the daily press and the reports of quacks, chiropractors, osteopaths, all quick to influence the lay mind to their own financial advantage, concerning a new and easy way of removing tonsils. No wonder the well trained otolaryngologist of high standing shows prejudice.

Even if these irregular men have almost made a racket out of the tonsils because the laity loves a novelty, there is no reason why conservative laryngologists should frown upon diathermy. On the contrary, they should take it up with an open mind and be able to use it for what it is worth.

Salesmen will make nonessential, irrelevant and exaggerated statements as to what their apparatus will do. When skillful otolaryn-

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gologists condemn diathermy because they do not immediately get the results they expect, it is usually because they will not take the time to study the apparatus and the effects of the different currents. When you see gullible doctors with little or no surgical training, who believe the statements made by the high pressure salesman, that all they need to do is to buy their apparatus, then go ahead and do their own tonsils, skin lesions, hemorrhoids, etc., it is no wonder that diathermy is in such disrepute, and the advantages of diathermy for certain well defined fields, as in otolaryngology, is obscured.

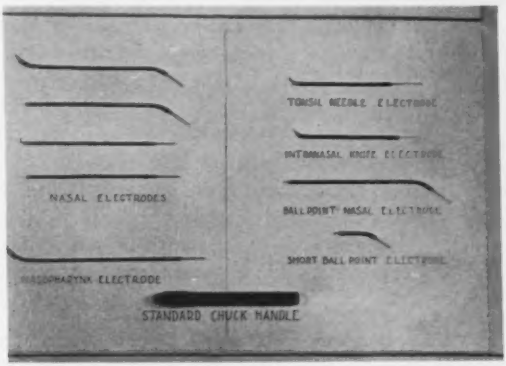

Fig. 1. Universal handle with tips. Any number of different shaped tips may be homemade to fit this handle.

When I took up diathermy seriously, I found that except for a few needles for tonsillar coagulation, there were no instruments applicable to nose and throat work, also that the manufacturers thought that a great deal more current was needed than was actually the case, which was the reason for their clumsy, thick, insulated instruments. The indifferent or dispersive electrode need not be a large, clumsy metal plate placed on the back or neck of the patient. The nasal speculum, tongue depressor, tonsillar retractor[1] or laryngeal mirror can take its place. In this way much less current is required and the patient is not apprehensive about being connected to an electrical apparatus. The back plate is used only when there is extensive cutting or coagulating to be done, under general anesthesia.

Anesthesia: Procedures with high frequency currents are painful, and whatever method of anesthesia you use in surgical operations

may be applied to these procedures. Personally, I have largely given up cocain since the admirable work that the late Dr. Emil Mayer's committee did on local anesthetics, and use alypin topically, and novocain hypodermically. I use alypin, 10 per cent to 30 per cent, plus a small quantity of adrenalin, in the nose, and a solution of equal parts alypin, analin oil and alcohol, in the throat. For example, to tonsil remnants, a small brush of cotton on an applicator moistened with two or three drops of the solution, is wiped on the area three times, at intervals of from five to ten minutes, and this usually produces a good anesthesia. Novocain by hypodermic works well, if you allow time for the edema to subside; if not, the radiating heat may boil the novocain and cause greater destruction than is intended. General anesthesia is necessary in the nasopharynx, usually with adults, always with children. The usual gas-ether sequence is given until we are ready to use the diathermy. Then the ether is stopped, the ether apparatus removed, and the patient allowed to come out until the reflexes are active. Anesthesia is then continued with chloroform for such time as the diathermy is in use. It is most important to have the ether and the ether apparatus removed from the table. The danger is that the mixture of ether and air may be exploded by an electric spark from either the transformer, the exposed spark gap, or about the electrode point itself.

The cutting current is a biterminal one of undamped high frequency, which divides the soft tissues by instantaneous cellular disintegration, with a searing of the cut surfaces to a depth varying with the setting of the diathermy apparatus, from a fraction of a millimeter to a deep coagulation. To date, the pure cutting current is only produced to perfection by the vacuum tube machines, and must be an entirely separate apparatus. The multiple spark gap apparatus produces a satisfactory current, but not a pure undamped wave; this is produced by crowding together the damped wave trains, and not allowing the oscillating potential to reach zero.

The coagulating or d'Arsonval current is a biterminal damped current of less frequency than the knife current, which destroys the tissues by coagulation. The speed of its action and the depth of the coagulation depend upon the strength of the current applied, the time it is allowed to act, the nature of the tissues being coagulated, and the size of the electrode used. The alternating current, as you know, is graphed as a wave that curves above the line, then down to zero, then curved below the line back to zero. One such double curve is called a cycle when produced in one second. The

regular street current, called low frequency, is sixty cycles to the second.

d'Arsonval found that by increasing the frequency, the muscular contractions increased up to 3,000 cycles and remained stationary between 3,000 and 5,000 cycles, then the contractions decreased up to 10,000 cycles. Now, with the oscillations around a million, there should be no neuromuscular effect. The high frequency current passing through the tissues creates heat by the resistance of the tissues to the passage of the current itself.

The heat can be regulated from mildly warm when passed between electrode plates of large area, to intense and destructive heat when one electrode is concentrated to a small area, such as a needle point, causing gross and microscopical changes in the tissue about the needle. One first sees a blanching, then as the current continues, the area turns white, then chars, and finally becomes black. The process is sometimes accompanied by the explosion of steam bubbles from the boiling of the liquid in the tissues. In practical uses the desired effect is reached when the blanching appears. There may also be some electrobiochemical phenomena. This is different from all previous known methods of applying destructive heat, such as the electrocautery and heated metals.

The desiccating or Oudin current is a monoterminal current of very high voltage, and of a frequency between that of the other two types of current, which produces a superficial dessication of soft tissue by drying and shrinkage. My experience has been that the coagulating current can be made to take the place of this current since it can be controlled with greater precision, and therefore I have practically abandoned use of the Oudin.

Uses: I will speak mainly of conditions wherein I have found diathermy superior or equal to the older methods. Also of the simpler conditions which I have personally evaluated. I will let the men who are trying diathermy in other conditions speak for themselves. Diathermy in malignancy, brain abscess, etc., has a very definite place, which I shall only mention. The novice has no right to venture into these fields.

The cutting current, used through a fine sewing needle to open abscesses, seals the cut surfaces and prevents extension of infection. To remove cysts, a small buttonhole, or a flap, is made, depending on the size of the cyst, the cyst contents evacuated and the lining membrane destroyed with the ball-tipped electrode, using a light

coagulating current. This method is as far ahead of the old surgical methods as the automobile is ahead of the oxcart.

For new growths, this method is fairly well established in general surgery. The malignant mass can be destroyed in situ, with the coagulating current, then removed with the cutting current without disseminating the malignant cells. There are two precautions to be observed; if coagulating near the bone, one must be prepared for superficial necrosis that will take some time to slough away, and hemorrhage when the slough detaches, if the large vessels have not been ligated.

The insulated snare is used for removal of nasal mucosal hypertrophies, without hemorrhage. The snare cannula is insulated, and fine steel piano wire used, from No. 1 to No. 3, since the ordinary No. 5 wire requires too much current.

The insulated snare for removal of tonsils in capsule is a combined Sluder and snare, modeled upon the Braun-Sluder snare, and devised by my associate, Dr. Morrison. The snare wire is charged with a cutting and coagulating current that sears the fossa, preventing primary hemorrhage from the small vessels, but does not always seal the large vessels. This has to be done with coagulating forceps, or by ligation. This method of tonsil removal is still in the experimental stage, and we are not prepared to state whether or not it has sufficient advantage over the standard practice to be practical. It requires chloroform when general anesthesia is used, and we have not as yet perfected the combined cutting and coagulating currents to a point where we can seal all the vessels to make the procedure bloodless, without causing too deep coagulation of the fossa. Also the fossae are about twice as long in healing. There can be secondary hemorrhages from the fifth to the fifteenth day.

The coagulating current is used for destruction of postoperative nasal granulations. For this work, special insulated nasal electrodes, with sharp or dull points, or ball tip, and straight or curved, are used, the nasal speculum acting as the dispersive electrode. Coagulation removes these granulations with greater efficiency and comfort to the patient, and there is no bloody discharge afterward.

Mucosal hypertrophies of the inferior turbinate are reduced with the double needle electrode.[2] The needles are of spring brass or steel, 5 cm. in length, and separated from each other by 3 mm., and slightly approaching each other at the points. Each needle is insulated from the other, and each is connected to one of the terminals

of the generator by a cable. In use the needles are pushed into the mucosal hypertrophy of the turbinate parallel to the long axis, avoiding contact with the bone, and well under the surface of the mucous membrane. The depth of penetration, and the number of coagulations naturally depend on the extent of the hypertrophy.

When the double needle is in place, the current is turned on and the coagulation starts at the needle points and works between the needles as a thin ribbon of coagulation in the submucous tissue, until it arrives at their point of entrance, which is indicated by a thin line of coagulation between the needles. Then the current is shut off and the needles withdrawn. This takes from three to five seconds. There should be no coagulation of surface mucosa except between the needles at the point of entrance. The hypertrophy usually shrinks at once, and there is no observable reaction, except at the point of entrance, where a small crust forms. This method is far superior to electrocautery or chemical caustics.

Nasal Synechiae: The older methods of cutting or cauterizing these usually resulted in prompt recurrence. Electrocoagulation is a great improvement, in that when the band is coagulated and the coagulum left in place, it acts as a barrier to reformation, while the two sides are healing.

Tonsil and adenoid remnants, and pharyngeal lymphoid follicles: Removal of these scattered masses of lymphoid tissue can be better accomplished by electrocoagulation than by any of the former methods. In adults it can be done under local anesthesia without interruption of the patient's daily routine, in from one to several sittings. In children it is done at one time under general anesthesia (chloroform). The appropriate needle is plunged into the lymphoid tissue, after first testing it on one small area to determine the setting of the apparatus. The area around the electrode point should blanch in two to three seconds, to a radius of 2 to 3 mm., depending upon the size of the point; the coagulated area will extend inward the same distance beyond the point. This is repeated until all the area is covered with coagulum. It takes about two weeks before the coagulum and slough entirely disappears.

Lymphoid tissue or adhesions in the pharyngeal recess (fossa of Rosenmuller), and about the Eustachian tubes, is a condition which is frequently overlooked and can cause recurrent acute catarrhal otitis media, and acute suppurative otitis media and tinnitus. The late Yankauer, of New York, and Holmes, of Boston, devised meth-

ods of cutting these out, but they usually failed because of the resulting adhesions, while with electrocoagulation there is less tendency to form adhesions. With the patient under chloroform, the area is gently inspected through a Yankauer nasopharyngoscope, taking care not to tear any of the adhesions and thus cause bleeding. As the bands of lymphoid tissue appear in the scope, they are destroyed by repeated application of the current, using various special electrodes, until all lymphoid tissue and adhesions are coagulated. Care must be taken in coagulating the lymphoid tissue below the Eustachian tubes or the result may be a paralysis of the levator veli palati muscle. With extensive lymphoid masses in deep pharyngeal recesses, we attempt to obliterate the recess. Great care must be used because of the proximity of bone and cartilage, both of which are affected badly by heat. Also because you have only the one opportunity as patients object to repeated general anesthesias.

For destruction of hypertrophied lingual tonsils and varicose lingual veins an electrode with a sharp point, 8 mm. long, slightly bent toward the lingual tonsil so that it can be plunged in obliquely, is used, with the laryngeal mirror as the dispersive electrode. In some cases of hypertrophied lingual tonsils, because of their shape, the guillotine will not engage the mass, but electrocoagulation will reduce these very satisfactorily. Varicose veins can be obliterated by touching each vein with the electrode point. This is equal but not superior to the electrocautery. Incidentally, I have entirely discarded the electrocautery in favor of diathermy.

Leukoplakia, when necessary, can best be treated with diathermy. The irritated patches can be gone over lightly with a ball tip applicator, using a weak coagulating current, just destroying the white membrane and not burning the submucosa. In indurated patches, deeper burning is required.

Removal of faucial tonsils: There is a great furor about this method. It has been taken up principally by men without connections, and I regret to state that certain manufacturers of apparatus have financed some of these men to do tonsils with their machines. I have read articles syndicated in the daily papers advocating this method, and stating that standard surgical methods are not only out of date, but criminal. I know of chiropractors who gather in patients for some itinerant burning artist, who comes around at regular intervals to burn each and everyone at so much per. Reputable men tell me that they have to do tonsil diathermy, or their patients will go to these irregular men.

Their selling talk, as you probably know, is that it is the only safe method, with very slight local discomfort, no interruption of the patient's daily routine, no hemorrhage, no infection, no lung abscess, in fact that it is the perfect method. They state that the tonsil is sterilized after the first or second burn and that the patient promptly feels generally better. They state that after doing hundreds, they have at last arrived at the perfect method. Some use a single needle, and some a double, some use all three currents, and others, one or two. Another statement made is that after a portion of the tonsil has been coagulated, they spray it with the Oudin current, then puncture the tonsil with the needle and use the Oudin to sterilize it and obviate after pain. The slight superficial dessication caused by Oudin sparks does little good, and since the Oudin spark has little effect without an air gap, when the needle is plunged into the tissue there is no effect whatever.

Let us take up each one of their arguments in order. First, pain. There is some discomfort for a day or two, depending on the temperament of the patient and the size of the burn. There is usually no interruption of the patient's routine. There is no hemorrhage at the time of the treatment, but when the slough begins to come away, there can be a secondary hemorrhage, which may occur any time from the fifth to the fourteenth day. It is quite true that as a rule this is not usually serious, though very severe hemorrhages have been reported, and at least it is annoying. As for infection, Dr. Morrison has had one case of acute cervical adenitis within twenty-four hours after the burning. No lung abscess has been reported to date. There is no logical reason why a burn of a portion of the tonsil should sterilize the balance of the tonsil; in fact, it does not do so, and the attached slough is far from sterile. Some men had the assurance to state that if part of the tonsil is burned out, the rest is clean and healthy. This is a good alibi for not doing a thorough job.

Electrocoagulation does not and should not take the place of good surgery. It has its place, however, in competent hands. In cases which are a poor surgical risk, such as patients who are elderly, patients with diabetes, or arteriosclerosis with advanced kidney and heart lesions. These men, to whom I referred above, also speak of hemophilia. I wonder if they ever used coagu'ation on a true case. I certainly would not use diathermy on such a patient any more than surgery, because when the slough detached they would probably bleed uncontrollably.

The technique is to use electrocoagulation with either a single or double needle, about 3 mm. in length. This is plunged into the tonsil and the current turned on, about five punctures at each sitting, doing one tonsil at a time. The interval between treatments to the same tonsil is at least two weeks, in some cases longer. The procedure is easy until you reach the lymphoid tissue behind the anterior pillar and in the superior fossa, where it takes considerable skill to remove this without burning through the capsule. The result should be a perfect tonsillar fossa. The drawbacks are: the time consumed, three to four months, requiring eight to twelve treatments to each tonsil, some pain or discomfort each time, and the possibility of an annoying secondary hemorrhage when least expected.

To control postoperative tonsillar hemorrhage, primary or secondary, I have been using the tongue depressor or tonsil retractor for the dispersive electrode, and an alligator nasal forceps with the shaft insulated, to pick up the bleeding points. In a tonsillectomy under local anesthesia, it is quicker than clamping and tying, and I have observed that the small points of slough remain for several days after the regular surgical slough has disappeared. I have not used this method with general anesthesia because of the time it would require to switch from ether to chloroform, and the fact that ligatures are just as safe, if not more so. I have had no secondary tonsillar hemorrhages on which to try it, except in cases where diathermy has been used, and then it seems that electrocoagulation is the best way to stop them.

MEDICAL DIATHERMY.

We define Medical Diathermy as the application of high frequency current to the body to produce warming of the tissues within the physiological limits. The effect is deep heat, occurring from without, inward. It is maximum where the current density is greatest, therefore a smaller electrode is placed over the area to be treated, and a larger dispersive electrode opposite. Besides the heat there is a hyperemia, hyperlymphemia and a decided analgesic sedative effect, and increased local metabolism.

Uses: In the ear, it is used for acute catarrhal otitis media, otitis serosa and furuncles, and we have found that pain is relieved, and drainage and resolution aided. It is applied to the external auditory meatus by a metal ear electrode, and the dispersive pad is applied to the opposite cheek. The heat is slowly raised to the point of tolerance, and maintained for about six minutes. If continued for

a longer period of time, the heat stimulates the labyrinth, causing vertigo, nystagmus and nausea.

For pain in acute sinusitis, after the usual procedures to promote drainage, diathermic heat is applied over the frontal or antrum, as the case may be, by means of flat sponge rubber electrodes containing a piece of block tin, so that they are flexible enough to fit accurately, and yet stiff enough to maintain their shape. The current is carried to the skin through a wire mesh on the surface of the sponge rubber, covered with a piece of moist tissue paper. A large dispersive electrode is applied to the back of the neck and held in place manually or by an elastic head band. The current is slowly increased until the heat tolerance is reached, and maintained for from fifteen to thirty minutes, once or twice a day. There should be no Faradic or sparking. The patient controls the current with the foot switch, so that it can be stopped immediately if it becomes too hot. This is the most satisfactory method of relieving this pain that I have found.

In conclusion, let me state that I feel that diathermy should be used only by experienced surgeons who know the anatomy and pathology of the region wherein they propose to use it. One must also know how to properly control the currents, and it is essential to practice on meat to determine the output and quality of the apparatus at different settings of the controls, before attempting its use on the living. It must also be remembered that living tissue may be devitalized beyond the visible area because of the penetrating heat, and that the use of the same electrode with a heavy current for a shorter time will not penetrate as far as a lighter current for a longer time.

I have mentioned only those conditions in which I have found diathermy to be superior or equal to any other method. I am sure that anyone who will learn the physics and physiology of the action of high frequency currents and apply this knowledge in a rational way will find its use a most helpful addition to the armamentarium of the otolaryngologist.

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39 East Fiftieth Street.

UNILATERAL PARALYSIS OF FACE, PALATE AND LARYNX. A CASE FOR DIAGNOSIS.*

DR. M. FELDMAN, Brooklyn.

Patient H. H., male, age 14 years, came to the clinic on Jan. 7 last, complaining of hoarseness for the past two weeks, regurgitation of fluid through the nose for about one week's duration, and disfigurement of face, noticed day before consulting us. Examination revealed complete (abduction) paralysis of right vocal cord, palate and lower part of face. The eye closed tolerably well and forehead wrinkled at patient's will. Two days later, however, the facial palsy was complete.

Family history essentially negative. Patient had convulsions during the first week of his life, with a temperature of 105°. The doctor saw blisters in the mouth, which he treated by application of medicine. Had measles and twice mumps; no diphtheria or any other illnesses.

Blood: Wassermann and Kahn negative. *X-ray Report:* The chest shows a small calcified lymph node in the left hilus, otherwise negative. The neurologist, after describing the various tests the patient had been subjected to, gave no hint as to possible cause of the disturbance.

I report this case, it being the first of the kind in my experience, with the hope that some in this audience may have come across a similar case, either personally or in the literature, who could throw some light upon the question of diagnosis. I personally have a suggestion, or rather ask, is it not possible that we were dealing here with a case of anterior poliomyelitis?

From the history and our observation, it is quite apparent that the larynx was first to be affected, the hoarseness having appeared first in this syndrome; the regurgitation of fluid through the nose occurring about a week later, would lead one to the conclusion that the palate was next attacked; and since the facial invasion took place while we were watching, it goes without saying that this was the last stage of the performance. The nerves most likely involved were the seventh and tenth cranial.

We have to take it for granted that all the three regions had been exposed at the same time. How to account for the gradual appearance of symptoms, first in the larynx, next the palate, and last the

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face, about a week apart from each other? For want of a better explanation, may I offer the following hypothesis: Just as not all the nerves function alike; just as motor nerves may give off sensory branches or filaments, which may likewise be said of sensory nerves; I can conceive of a possibility, unless there is proof to the contrary, that not every nerve is possessed of the same amount of affinity for the specific poison. Clearly, to my mind, it is possible that of the nerves involved, the pneumogastric, or rather the superior and recurrent laryngeal branches thereof, contain the most of the chemical which is responsible for the attraction, and, therefore, the laryngeal symptoms manifested themselves earliest. The branches of the facial supplying the levator palati and azygos uvulae muscles contain less, and those distributed to the muscles of expression the least, and for that same reason the symptoms followed the above mentioned order.

We observed a very interesting phenomenon watching the course of the recovery. Its order bore a striking similarity to that of the invasion, but in the reverse direction. The face, last paralyzed, cleared up first. When the face appreciably improved, the palate showed but a slight change to the better; and when the face, to all appearances, was restored to normalcy, there was still quite some dysfunction distinctly visible in the palate, which completely disappeared from the picture several days later. The fate of the larynx, however, was not such a lucky one, the cord having remained immovably fixed in the median line without any improvement whatever.

What explanation can possibly be advanced for this? I am unable to think of any other than the following: Soon after the face had been completely paralyzed, there accumulated enough of antitoxin, or call it what you may, to start the repair. The nerves concerned in the facial palsy, having suffered in the mildest degree and for the shortest time, recovered first; those supplying the palate having been under the influence of a more intense attack and for a longer time, having thus sustained more injury, more damage, required more time for repair. And, finally, applying the same reasoning to the laryngeal nerves, it would seem that because of the reaction having been too severe and the duration too long to endure, they have been hopelessly damaged beyond repair.

In conclusion, I wish to state that, although the patient was only fourteen, his weight tipped the scale at 214 pounds and, what is of real importance, his blood pressure was 200, and the pulse ranged between 100 and 160. However, this probably belongs rather in the domain of endocrinology, and should have no bearing upon the subject we are concerned with.

1034 St. Johns Place.

SEPTIC SORE THROAT.*

Dr. Calvin G. Page, Boston.

The epidemic in Boston, Brookline and Cambridge in May, 1911, was the first in this country to be designated as septic sore throat.[1] In England there had been several large epidemics under that name.[2]

The sudden onset, the peculiar reddening and infiltration of the pharyngeal wall, as well as of the tonsil, the marked prostration and tendency to relapse, the slow pulse, irregular temperature and other peculiar clinical features marked this as an unusual condition. There were a number of fatal cases of peritonitis. The high mortality in people over 60 years, 31 per cent, with 11 per cent in those of 40 to 60, marked it as a serious condition.

Professor Sedgwick's quotation from a private letter is worthy of repetition.[3] "The death of Mrs. X seems the very culmination of this tragic season. Cambridge is like a city of the plague. Whomever one meets, the talk is all of who is ill, who has had a relapse, who has died. The diagnosis of it is ill; it is no tonsillitis, but a most malignant poison, issuing now in erysipelas, now in abscesses, now in rheumatism or neuritis; sometimes fatal in three days. All of the principal doctors here are convinced that it comes through milk."

The next year there was an epidemic of 10,000 cases in Chicago,[4, 5] with a mortality of over three per cent. These epidemics and others[6, 7, 8, 9, 10, 11] illustrate very well the non-use, the breakdown and the improper operation of milk pasteurizing plants.

"Streptococcus epidemicus" is the name attached to the organism isolated by Dr. David J. Davis from cases of septic sore throat in the Chicago epidemic of 1912.[14] He examined four cultures from the Boston epidemic and found them the same as those from Chicago. The name has been used tentatively by several workers with organisms from similar epidemics.[6, 13] These have been traced to

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*From the Otolaryngological Laboratory, Massachusetts Eye and Ear Infirmary.

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a cow with one teat infected by a milker who had septic sore throat.[15, 16, 17, 18, 19, 20, 21, 29, 30, 31]

There are two characteristics of this variety of streptococcus hemolyticus which were supposed to differentiate it clearly from other hemolytic streptococci. Recent work by Dr. Anna Williams[12] and associates shows that neither of these two characteristics belong exclusively to this type of hemolytic streptococcus and that therefore "Streptococcus epidemicus" is not permissible as a species name, but may be used tentatively for convenience.

The first characteristic claimed for S. epidemicus is that on fresh moist blood-agar pour-plates, especially if 2 cc. of ascitic fluid is added to each tube of agar before pouring—the colonies are moist, raised and larger than the usual hemolytic colonies and that the surface colonies soon flatten out.

The addition of ascitic fluid was suggested by Dr. Isadore Pilot[22] of Chicago. Others have observed the same appearance of the colonies if the air in the incubator is kept saturated with moisture. Dr. Williams claims to have seen large moist colonies in several different types of hemolytic streptococci.

The other characteristic is the appearance of a capsule or capsular space along the chain of cocci from recently isolated cultures when observed in moist india ink preparations. Dr. Williams shows photographs of three other types of hemolytic streptococci which show distinct capsules. She records a good method for making stained preparations of cocci with capsules.[12] Dr. Davis claims that animal passage will restore the formation of capsules by a culture if that character has been lost.

Dr. Pilot has written a number of short articles in the *Journal of Infectious Diseases*[20-28] in regard to septic sore throat. He claims to have cultivated this streptococcus in 13 per cent of tonsils excised from 281 patients. I thought it would be interesting and important to know if any such percentage could be found in Boston. From October 13, 1931, to July 27, 1932, I made cultures of tonsils excised from 200 patients. At the operation the tonsils were placed in sterile bottles and stored in an ordinary ice chest until the next morning. They were then dipped in alcohol, flamed and cut with a knife similarly sterilized. Loopfuls from the center or a crypt were transferred to a tube of melted agar cooled to 50° C., to which had been added 0.5 cc. defibrinated horse blood and 2 cc. of ascitic fluid. The tube was rotated and the contents poured into a sterile Petri dish. Sometimes a dilution was made by transferring a loopful from the

tube just before pouring to a similar tube of agar. At other times inoculation from the tonsils was made into a tube of broth and then the agar inoculated with a loopful of the suspension. The broth tubes were incubated along with the plates and examined the next day and the following day.

In none of the 200 cultures did I find hemolytic colonies with moist surfaces that flattened out on the second day and which showed a capsule in moist India ink preparations. Dr. Pilot sent me a culture marked Type C which I used for comparison. He also sent me 10 cc. of a 1 to 100 dilution of the filtrate of this culture to be used for skin tests. I inoculated eleven individuals, nine positive, two negative. I immunized myself and one of the house officers at the Massachusetts Eye and Ear Infirmary. I found it necessary to dilute the filtrate still further to 1 to 500. I immunized a rabbit with twelve doses of the filtrate between November 5th and January 14th. The rabbit serum gave a positive agglutination test and a positive complement fixation test using the active complement[32] in the rabbit's serum. On the following day a repetition of the test with the same materials showed only a slight loss of complement.

In clinical cases of septic sore throat the infection may extend to the middle ear and mastoid. It may cause cellulitis and deep abscesses of the neck. If the tonsils have been removed the organisms do not remain in the throat beyond convalescence.

There are carriers of this organism, but tonsillectomy is the accepted treatment for that condition.

In some milk-borne epidemics, scarlet fever has been the chief recognized condition, with a few cases of sore throat without rash. In other epidemics sore throat has been predominant, with few cases showing rash or desquamation. In any epidemic it is difficult to be certain of isolating the causative organism because for one thing there are other types of streptococci in the victims' throats and in the milk. The best method now known of completely identifying different types of streptococci is by the agglutination test and the absorption of agglutinins and cross absorptions. Dr. Williams finds that there are four agglutinative groups of scarlet fever streptococci and one sub-group. For erysipelas there are two groups and two sub-groups. For septic sore throat there are two groups, one of which is closely related to one of the sub-groups for erysipelas.

In future epidemics, Dr. Williams thinks it would be well to test the children, and adults, too, with standard scarlet fever toxin to see

if they were already immune. Also to test with erysipelas type toxin and with "epidemicus" type toxin.

In most milk-borne epidemics there have been comparatively few "contacts," i.e., cases where the infection is transferred from one human to another. The proportion has been estimated at not over five per cent. Nevertheless, as a preparation for a future possible epidemic, would it not be advisable for some nurses and physicians to have skin tests made in order to learn to which types of streptococcus infection they were susceptible? If they so desired they could be immunized by five or six minute doses of toxin, repeated at intervals of one week.

It remains for future efforts to produce an effective anti-serum for treatments of "epidemicus" infections, but meantime milk-borne epidemics can be prevented by efficient pasteurization of raw milk.

SUMMARY.

A brief outline of milk-borne epidemics of septic sore throat is presented and the two characteristics by which the "epidemicus" type of S. hemolyticus has been recognized.

Report of examination of tonsils excised from 200 patients in Boston with negative findings as to streptococci with these two characteristics.

Reference to work of Dr. Anna Williams on identification of hemolytic streptococci by agglutinin absorption tests.

Suggestion that skin tests of patients and also of nurses and doctors be made to determine immunity or susceptibility, with the possibility of immunization by multiple doses of toxin.

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128 Marlborough Street.

TEACHING TONSILLECTOMY.*

Dr. Joseph D. Seiberling, Philadelphia.

Certain pernicious tendencies, insidious in their development, have pervaded the operation, tonsillectomy. Constructive reverberation, figuratively speaking, is in order. In the following generalized discussion, the scope includes the preceptor or teacher, the pupil and the surgical or technical art. A heterogenous triad—the consociation of which may make for excellence in its achievement or be a vicious cycle, the ulterior motive culminating in its derogative influence upon the favorable merit of tonsillectomy.

One privileged to teach a technical surgical procedure has assumed responsibility of momentous significance. A responsibility measured in part by previous instruction and experience, but above all, by conscience. The preceptor, a costudent, is the product of the effort of one gone before. His far-reaching influence upon his pupil at once becomes apparent. He must of necessity be possessed with several cardinal qualifications, namely, meticulous attention to detail—rigid adherence to the principles of moral responsibility and persistent adherence to the practice of asepsis.

Meticulous Attention to Detail: Surmounting in significance is deportment. It remains the unqualified duty of the teacher to impress his pupil with sincerity of purpose, promote profound regard for the dignity and solemnity of the occasion and to stimulate aspiration to lofty ideals.

Rigid Adherence to the Principles of Moral Responsibility: First as it concerns the case, and second the student. Exhaustive study of each case as an entity, arriving at definite conclusions for surgical operation. The writer, wishing to emphasize only the qualifications of the tonsillectomist, will omit the details implied in exhaustive study of each case. To the student must be conveyed the extreme importance of the preoperative study of the case, as referred to previously, but especially as it applies to a careful history, the significance of which is revealed in a report by Collis[1] of two fatal cases of septicemia following tonsillectomy as a result of unrecognized exposure to scarlet fever. Briefly then, it behooves the teacher of tonsillectomy, by his demeanor, attention to details and surgical vision, to

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perpetuate profound regard for the moral and technical qualifications of those who contemplate operative surgery in the pharynx.

Persistent Adherence to the Practice of Asepsis: In no field of operative surgery is the danger of carrying infection greater than it is in the removal of faucial and pharyngeal tonsils. Ide[2] reports a case of cavernous sinus thrombosis following tonsillectomy, and Comer[3] encountered a similar experience. It is incumbent on the teacher to be referring repeatedly to the immediate and remote potential dangers of tonsillectomy, thereby inculcating mutual regard for the effort to avoid those dangers.

Quoting Chevalier Jackson[4] relative to teaching bronchoscopy and esophagoscopy, his preliminary remarks may as well include tonsillectomy: "When one considers the highly technical nature of bronchoscopy and esophagoscopy, the lack of literature is remarkable, and when one considers that fully half of the patients requiring these procedures are infants, the lack is deplorable. It becomes utterly so when it is realized that the lack leads the thoughtless to infer that training is not necessary."

The Pupil: In the preface to the first edition of "Diseases of the Nose and Throat," St. Clair Thomson emphasizes the major qualification of the prospective student of tonsillectomy, by the following: "The experience gained during some years in general practice has, I trust, not only served me from taking too narrow and too mechanical a view of the disease of the air passages, but has also helped me to realize their increasing importance in every-day work and enabled me to supply such information and assistance as a practitioner is likely to require." The aphorism, putting the cart before the horse, has no more appropriate portrayal than that depicted, when those unqualified in "some years of general practice" are bold to perform tonsillectomy. "Too narrow and too mechanical a view" before one may intelligently grasp the importance of tonsillectomy, especially the indications for and the therapeutic results expected, he must have broadened his vision and have developed a keen sense of evaluation. Presuming that the prospective student has attained this general knowledge in extensive general practice, he is now in a position to show cause why he should not elaborate on the qualifications required of the well trained tonsillectomist.

This brings us to the mechanical or technical art. Unless the student is cognizant of the skill and dexterity required in the surgical manipulations in the pharynx, he is lucky indeed not to reach for a machine instead of instruments of precision. Quoting Jackson,[4] "The

marvelous hand-forging of the expert iron worker requires as much manual skill as the making of the old handmade Swiss watch, but the latter requires a different type of skill, possessed by a different type of man. The heavy handed man is not the most promising raw material from which to make the safe bronchoscopist." This may as well include the tonsillectomist.

Operating with a microscopical eye and intimate knowledge of the surgical anatomy of the pharynx will foster a desire to attain supremacy in the refinement of this very important surgical procedure. He, therefore, who visualizes the mucous membrane as a highly specialized organ, performing a specific physiological function and is aware of the morbid processes following its injury, will not include it in tonsillectomy. Similarly the concern one must have for blood vessels, nerves and muscles contiguous to the tonsils implies a detailed knowledge of histology. This is true especially in the effort to avoid lacerating fibroareolar tissue, thereby opening avenues of infection. Too often is the pharnygeal tonsil attacked blindly. A careful pre- and postoperative study of the surgical anatomy in relation to it will avoid errors of commission and omission.

Finally the future tonsillectomist must first of all be a proficient assistant. That qualification can only be acquired by a term of apprenticeship. This latter requirement is looked upon with deplorable indifference by the beginner in operative surgery, rather assuming it to be an obligation incumbent on the artisan. Durtrow[5] in conclusion to his paper remarks "that refinement in operative technique may be obtained by study and observation of the work of skilled and experienced laryngologists." Indeed, until the state demands indenture apprenticeship of the tonsillectomist, provided the medical profession fails to take the initiative, otolaryngology, an otherwise healthful vocation, remains severely blighted.

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1737 Chestnut Street.

LATERAL SINUS THROMBO-PHLEBITIS WITH EXTENSION TO THE TORCULAR; REPORT OF TWO CASES; OPERATION WITH RECOVERY.

DR. SAMUEL D. GREENFIELD, Brooklyn.

It is a notable fact that the structures about the lateral sinus may be extensively diseased, including the sinus wall itself. Clinically there may be neither sign nor symptom to indicate either the extent or the degree of such involvement. I am sure there are very few otologists, no matter how limited may be their experience, who have not in the course of their mastoid operations unexpectedly come upon a number of pathological processes in which the sinus wall has shared in no small measure.

There are various degrees of involvement which one may encounter. The sinus plate viewed macroscopically may appear normal in color, conformation and consistency. Its removal, however, either intentionally or accidentally, may disclose the existence beneath this structure of a number of pathological processes. On the other hand, the sinus plate may be found softened and necrosed and the lateral sinus exposed to a greater or lesser extent. The normal bluish hue of the sinus wall may be absent and the latter reddened, edematous and slightly thickened, with fine granulations on its surface. In other instances the sinus wall may lie exposed for a considerable extent, forming the inner limit of a smaller or larger abscess cavity: the sinus bathed in a pool of pus. The sinus wall may be covered with a mass of diseased granulations of varied thickness, soft in character, mushy in consistency and bluish-black in color. There is no doubt that in these extreme cases the infection has penetrated the sinus wall itself but has evidently stopped short of the interior of the sinus. One cannot help therefore but be struck by the lack of clinical findings and reactions in response to such apparent intense and extensive disease of intracranial structures.

A feature that is even more striking is the well established clinical observation that cases of this character usually develop no compli-

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cations and, contrary to expectation, manifest little or no postoperative reaction. Furthermore, these cases heal very readily and rapidly. It is for this reason that we have come to look upon the formation of these granulations as an expression of the patient's resistance and denotes a protective phenomenon. They serve as a barrier against invasion of the interior of the sinus by the infectious process. Therefore, the growing opinion that unnecessary handling of these granulations on the sinus wall is prohibited. Any attempt to remove them, especially with the curette, is absolutely precluded.

The practice of most otologists in cases of this kind is to remove the sinus plate well beyond the limits of the portion of the sinus that is affected. This as a rule is a simple matter. However, occasionally the involvement of the sinus wall extends upward well beyond the knee or downward beyond the tip in the direction of the bulb. In those cases in which the temperature is not of a septic nature and there are present no symptoms to indicate that the interior of the sinus is implicated I do not believe one is justified in attempting to uncover to the entire limit nor do I feel that this procedure is necessary. In these extensive cases going downward in the direction of the bulb in an effort to reach the lowermost limits of the disease is not without danger. Likewise going backward to the torcular for the same purpose entails wholesale removal of too much bone. It is therefore my opinion, as I have practiced it in a number of cases of this type, to remove a goodly portion of the sinus plate without any attempt to uncover to the full limit. I do not recall a single instance in which a complication followed the employment of this procedure.

However, it is an entirely different matter where there are symptoms present which lead one to suspect the possibility of an extension to the interior of the sinus, as manifested by a temperature that is persistently high or of a septic character. It is certainly agreed by all that in these instances nothing short of exposure sufficiently wide and adequate to reach normal sinus wall in all directions is the only and proper surgical procedure. But even when this is done it should be borne in mind that what appears to be normal sinus wall externally may not correspond to healthy internal wall. With exposure to normal sinus the possibility of extension of disease beyond our operative field at a future date is of course not precluded.

The two cases herein reported present a number of features of unusual interest to which I wish to invite especial attention. In Case 1 we have a patient presenting symptoms of an acute surgical

mastoid. Before operation there was nothing to point to the presence of a perisinus abscess and certainly neither sign nor symptom to indicate involvement of the sinus wall itself. At operation I found a large perisinus abscess; the sinus bathed in a pool of pus; its wall markedly thickened and covered with an abundance of soft, mushy granulations. At one point these granulations were necrotic and black. The sinus plate was removed widely in all directions until what appeared to be normal sinus wall was visible in the circumference of the diseased area. Following the mastoidectomy the patient had a normal postoperative course and after being afebrile four days he was discharged from the hospital.

Two weeks later, when the posterior wound was almost closed and the middle ear was resolved the patient had a chill, which was followed by a quick rise in temperature to 105°. He was readmitted to the hospital and a blood culture was taken immediately upon admission. The next morning it was reported positive for the streptococcus hemolyticus. Recalling the pathological findings at the primary mastoid operation there was no question as to the diagnosis. I tied the jugular vein and opened and drained the lateral sinus, from which I removed a large well organized thrombus. The postoperative course was again very typical. He received the usual transfusions and general supportive treatment. At the end of four days his temperature was normal and remained so until the eighth day. Two blood cultures taken during this period were both returned negative. On the ninth day the patient again had a chill and his temperature rose to 105°. The blood cultures were again positive for the streptococcus hemolyticus.

This indicated the reformation and extension of the thrombus either to the bulbar or torcular end. I again opened the mastoid wound and found that the disease had extended backward to involve the torcular end. I uncovered the lateral sinus backward for a distance of two and one-half inches until I struck healthy sinus wall. I then incised and drained the exposed lateral sinus, at the same time removing a number of well organized thrombi. After this operation the patient went on to an uneventful recovery.

In Case 2 the patient's history was somewhat different. A diagnosis of sinus thrombophlebitis and bacteremia was made before the primary mastoid operation. There was a history of chills, a septic temperature, a recent acute otitis and finally a positive blood culture soon after admission to the hospital. At the time of operation the otoscopic picture revealed a resolving middle ear. I ligated the

jugular vein and found a badly broken down mastoid. There was a fistula in the lateral sinus from which free pus exuded. The sinus was uncovered widely and all thrombi removed. Free bleeding was obtained from the torcular end only. After a week of irregular temperature the blood cultures became negative and it appeared as if the patient was going on to an uneventful recovery. However, just as in the previous case, on the tenth day the patient had a chill, followed by a rise in temperature to 106°. The blood culture was again positive and the patient appeared gravely ill. Again the picture was one of a new thrombus formation and extension either backward or downward. I reopened the wound and as in the previous case found the extension had taken place in the torcular end. I removed cortex over the lateral sinus for a distance of three inches until I came upon healthy sinus wall. I removed some organized thrombi and slit the diseased portion of the sinus widely open. Free bleeding was obtained from the torcular end only. The patient then went on to an eventful recovery after ten days of a rather stormy course.

Case Reports: Case I: J. B., six years old, male, was admitted to the Beth Moses Hospital on March 10, 1930. His chief complaints were pain in the right ear of three weeks duration, temperature and aural discharge.

Previous Personal History: The patient had measles at three years. At four he had a tonsilloadenoidectomy. Despite this operation he was subject to frequent colds. At five years of age he had a bilateral suppurative otitis media at which time he had a bilateral myringotomy performed. After two weeks of otorrhea he cleared up.

History of Present Illness: Four weeks prior to admission the patient had a severe head cold. One week later he complained of pain in his right ear. Several days later the drum ruptured spontaneously. I saw the child in the third week of the otitis. At this time I found an exquisitely tender mastoid, a profuse aural discharge, marked canal changes and a temperature of 101°. I recommended operation.

Operation, March 10, 1930: The pathological findings were as follows: The mastoid was large and highly pneumatic. The cells had coalesced and were completely broken down. The sinus plate was destroyed over a considerable area. There was a large perisinus abscess. The sinus wall was covered with a thick coating of soft mushy granulations. At one point on a line with the mastoid antrum the granulations were discolored and black. I uncovered the sinus

for quite a distance and finally was able to visualize normal sinus above and below. The mastoid wound was brought together partially with two sutures and the wound drained. The pus from the wound was subsequently reported streptococcus hemolyticus.

The postoperative course was uneventful, as I have already intimated. The patient was afebrile on the third day and he left the hospital on March 16. He continued his dressings at my office apparently on the road to what appeared to be an uneventful recovery. On March 27, the patient complained of headache and had a temperature of 101°. The next day his temperature rose to 102°, and he appeared ill. He suffered anorexia and that evening he vomited twice. On March 30, the patient had a chill and his temperature rose to 104°. He was then admitted to the hospital on March 31, and a blood culture taken.

Admission Note: General physical examination is entirely negative. With a history of a recent mastoid operation, with the pathology noted at the mastoid operation, with the occurrence of chills and temperature, there is no question that this is a case of otitic sepsis.

April 1, 1930: Blood examination: Hemoglobin, 55 per cent; R.B.C., 3,200,000; W.B.C., 21,000; polys., 82 per cent; lymph., 18 per cent. The blood culture taken the day before was reported positive for the streptococcus hemolyticus.

Operation, April 1, 1930: The jugular vein was ligated in the usual manner above the facial and the neck wound closed and drained from the lower angle. The old mastoid scar was opened and the lateral sinus visualized. The latter was covered with an abundance of diseased granulations. The sinus plate was removed up to the knee and down to the tip. After plugging off the sinus the latter was incised and no bleeding obtained. The clot from the bulbar end was removed and free bleeding occurred. A large organized thrombus about one inch long was taken out of the torcular end and this was followed by moderate bleeding. A section of the sinus wall was excised and the sinus obliterated. The wound was packed with iodoform gauze held in place with one suture. The patient left the operating room in good condition.

April 2, 1930: The patient's general condition was fair. His temperature rose in one evening to 104.6°. He complained of little pain and seemed fairly comfortable. A transfusion of 300 cc. was given from a professional donor by the direct method. There were neither chills nor chilly sensations during the last twenty-four hours.

April 3, 4, 5, 6: The patient reacted favorably. For two days following the operation the temperature reached 104°. On the 5th it reached 103° and on the 6th it remained down. He received two transfusions each of 300 cc., one on the 3rd, and one on the 6th. Blood cultures taken on the 3rd and the 6th were both reported negative. R.B.C. on the 6th was 3,700,000 and hemoglobin 62 per cent.

First full dressing performed on the 6th under gas oxygen anesthesia. The plugs were removed from the sinus and no bleeding occurred. There was a welling up of pus from the bulbar end into which a drain was inserted. The neck wound healed by primary union and the sutures were removed.

April 7, 8, 9, 10, 1930: During these four days the patient gradually improved. His temperature was normal each morning and only on the 9th did it rise to 101.4°. The dressing was changed on alternate days and nothing unusual noted.

April 11, 1930: At three o'clock in the afternoon the patient had a violent chill which lasted twenty minutes. Soon the temperature rose and reached 106.4° at six o'clock in the evening. A blood culture was taken at the height of the temperature. A general physical examination was entirely negative. There were no neurological signs except a suggestive Kernig on the left side and some neck rigidity which appeared voluntary on the part of the patient. Nevertheless, a lumbar tap was performed and 15 cc. of clear spinal fluid under slight pressure were withdrawn. Examination of the fluid revealed nothing pathological.

April 12, 1930: The blood culture of the previous day was positive for the streptococcus hemolyticus, so there was no doubt as to what we were dealing with. Evidently there was extension of the phlebitis either toward the torcular or down to the bulb.

Operation April 12, 1930: The mastoid was again reopened and the sinus visualized. The drain from the bulbar end was removed and the latter seemed to be celan. The drainage of pus from this source had ceased several days before. The cortex over the sinus in the direction of the torcular was removed and the sinus found to be thickened and discolored. The bone for over a distance of three inches was bitten away and the lateral sinus layed bare. Its interior was entered and some organized thrombi were removed. The sinus was then slit open throughout its length. Some free bleeding was obtained. The wound was then packed and left widely open.

April 13, 1930: The patient seemed to be in much better condition than I had anticipated. The temperature rose in the evening to 105.2°. He was given a blood transfusion of 300 cc. by the direct method. Following the transfusion he had a chill and then a rise in temperature to 107.2°. This evidently was the direct result of the transfusion.

April 14, 15, 16, 17, 18, 19, 1930: During these six days there was nothing unusual noted. The temperature rose each evening, but the peak seemed to be lower with each successive day. There were no chills nor chilly sensations. The wound was dressed on alternate days. A blood culture taken on the fourteenth was reported negative after seventy-two hours.

April 20, 21, 1930: The patient's temperature reached normal on the 21st and remained so thereafter.

He was taken to the operating room and under gas oxygen anesthesia the margins of the wound were freed and brought together over the large gap in the scalp occasioned by the extensive removal of bone and laying bare of the sinus. This reduced materially the size of the wound and the granulating area.

The patient remained in the hospital another ten days, after which he was discharged well on the road to recovery. He continued his dressings at my offices and was completely healed by the 26th of May. He was discharged with a firm posterior scar and a dry middle ear.

Case 2: M. P., male, aged 8 years, was admitted to the Beth Moses Hospital on December 13, 1930, the chief complaint being severe pain in the head and a fluctuating temperature.

Previous Personal History: The child had measles at two and broncho-pneumonia at three years of age. At five he had a tonsillo-adenoidectomy.

History of Present Illness: Five weeks prior to admission the patient had a head cold. Several days later he began to complain of pain in his left ear. A physician saw him at that time and prescribed some ear drops. His pain abated somewhat for the next few days, but then he again suffered headache and spent many sleepless nights. Two weeks after the onset of his illness his temperature began to fluctuate, low in the mornings and up in the evenings. He developed some glands in his neck and the attending physician attributed his hyperpyrexia to his adenitis. He continued this course

until one week before admission, when a physician incised his drum. The ear discharged moderately, but the temperature continued. I saw him on the 13th of December and advised hospitalization for the purpose of observation and study.

Admission Note: Temperature, 103.6°; pulse, 133; respiration, 31. The general condition of the patient is fair. There is marked pallor and a coated tongue. The head is held in a retracted position, but there seems to be an indurated gland on the left side deep below the sternomastoid muscle. The heart and lungs are normal. The abdomen is negative. The extremities and skin are normal. There are no eruptions nor petechiae. There is no Babinski and no Brudzinski, but a suggestive Kernig and Tache on the left. Otological examination (Dr. Greenfield) discloses a scanty discharge in the left external auditory canal. There is a small perforation in the lower posterior part of the drum membrane, but the general appearance of the drum would be suggestive of a resolving otitis media. There is slight mastoid tenderness over the tip.

Conclusions: With a septic temperature, negative general physical examination and a recent history of an otitic infection, a diagnosis of an otitic sepsis is justifiable. However, before operative intervention I would suggest a blood culture, blood count, urine examination and with the suspicious neck rigidity a spinal tap should be performed.

December 14, 1930: Blood culture taken on the 13th was reported positive for the streptococcus hemolyticus. Blood count: Hemoglobin, 56 per cent; R.B.C., 2,800,000; W.B.C., 18,000; Polys., 82 per cent; Lymph., 18 per cent. Urine examination was negative. Spinal tap showed a clear fluid under normal pressure with 6 cells per c.mm. A preoperative diagnosis of thrombophlebitis of otitic origin was therefore made and operation advised.

Operation December 14, 1930: The mastoid was small and shallow of a sclerotic type. There were few cells here and there. The mastoid antrum was small and no free pus encountered. The few cells at the top were moderately softened. The sinus plate was unbroken and appeared normal. The sinus was exposed and found to be thickened edematous and discolored. Down in the direction of the tip there was found a localized collection of pus between the sinus and sinus plate. At this point the sinus wall was necrotic and a fistulous tract into which a probe could be introduced led into the interior of the sinus from which free pus exuded. The sinus wall was laid bare and the cavity temporarily packed. The jugular vein

was then ligated and the neck wound closed and drained from the lower angle. Returning to the mastoid wound a number of organized clots were removed and free bleeding occurred from the torcular but no bleeding could be obtained from the bulbar end. The sinus wall was excised and a drain inserted down in the direction of the bulb. The cavity was packed in the usual manner. The patient was immediately given a direct transfusion of 250 cc. and he left the operating room in fairly good condition.

December 15, 16, 17, 18, 1930: During these four postoperative days nothing unusual was noted. His general condition rapidly improved. The temperature each morning dropped to below 100°, but rose in the evenings. The highest rise was on the 15th, when it reached 104.4°. His dressing was changed on the afternoon of the 18th under gas oxygen anesthesia. The neck wound was clean and the sutures were removed. The mastoid cavity was fairly clean. The plugs were removed from the sinus and no bleeding occurred. The drain from the bulbar end was withdrawn. Following this one noted free pus welling up from below which pulsated freely. After cleansing the lower end another drain was introduced. The blood culture taken on the 17th was reported negative for the first twenty hours. Another culture was taken on the 18th.

December 19, 20, 1930: The patient's temperature on both these days dropped below 100° in the morning and rose to 102° in the evening. The blood cultures taken on the 17th and the 18th were both reported negative. Blood count: On the 19th, Hemoglobin, 62 per cent; R.B.C., 3,700,000; W.B.C., 11,000; Polys., 79 per cent; Lymph., 21 per cent. The patient received another transfusion of 250 cc. by the direct method the same day.

A full dressing was performed on the 20th. The mastoid cavity was clean. There occurred no bleeding from the sinus, but the bulbar end still drained pus, and a drain was reinserted.

December 21, 22, 23, 24, 1930: During these four days nothing unusual was noted. The temperature reached normal each morning and only on the 22nd did it rise to 101.2° in the evening. A dressing performed on the 22nd and 24th showed a disappearance of the pulsating pus from the bulbar end and the drain was no longer inserted. The patient seemed to improve rapidly and was apparently on the road to recovery.

At 4 P. M. on the 24th, the child had a violent chill, following by a quick rise in temperature to 105.2°. He complained of severe head-

ache and vomited three times during the afternoon. His neurological examination was entirely negative. The urine showed an occasional pus cell and some albumen. A blood culture was taken immediately after the rise in temperature. A blood count showed: R.B.C., 3,200,000; W.B.C., 22,000; Polys., 89 per cent; Lymph., 11 per cent.

Again it seemed that this patient had an extension of his thrombus beyond the operative field either downward to the bulb or backward in the direction of the torcular. The blood culture the next afternoon was reported positive for the streptococcus hemolyticus and the patient was reoperated the same evening.

Operation December 25, 1930, 9 P. M.: The mastoid wound was freed from granulations and the sinus visualized. The cortex was then removed in the direction of the torcular for a distance of two and one-half inches and the lateral sinus clearly outlined. The latter was thickened and distinctly diseased. The sinus wall was then slit open throughout its exposure and several thrombi removed. Moderate bleeding was obtained and after introducing a drain into the torcular end the wound was packed with iodoform gauze and a firm dressing applied. The child left the operating room in fairly good condition.

December 26, 27, 1930: The temperature in the morning of the 26th dropped to 102°, but rose again in the evening to 105.4°. He received a blood transfusion of 250 cc. His general condition was fair, but he seemed drowsy most of the day. His pulse rate varied between 140 and 160.

The patient's temperature in the morning of the 27th reached 101.4° and rose in the evening to 105.2°. He had no chills, but as on the previous day, he slept most of the time. His dressing was saturated with ser-sanguinous discharge and had to be changed twice that day. Hemoglobin was 50 per cent and R.B.C., 3,000,000. His pulse continued rapid and he received cardiac stimulation and forced fluids.

December 28, 29, 30, 31, 1930: On each of these days the temperature dropped in the morning and rose in the evening to above 104°. He received a transfusion on the 28th and the 31st. His pulse rate slowed up and despite his fluctuating temperature, his general condition improved. His full dressing was changed on the 30th. No bleeding occurred from the sinus and upon removal of his drain from the torcular, the sinus remained dry. A culture was taken on the 28th and was reported negative.

January 1, 2, 3, 4, 1931: During these four days the temperature fluctuated between 100° and 104°. His blood culture of December 31st was reported negative. Despite his fluctuating temperature his general condition remained fair. He showed no evidence of metastasis and had no complaints. He received a transfusion of 250 cc. on the 3rd of January.

January 5, 6, 7, 8, 9, 1931: During these five days the temperature remained normal and his general condition improved rapidly. He ate heartily and rapidly gained weight. It seemed now that he was well on his way to recovery. On the 9th the patient was taken to the operating room and under gas oxygen anesthesia the edges of the scalp were freed and brought together, thereby diminishing the size of the wound and the granulation area. The neck wound was completely healed.

The patient's condition thereafter rapidly improved and after remaining afebrile another week, he left the hospital on January 17, 1931. He was discharged on March 16, 1931, with a firmly healed posterior wound and a dry middle ear.

169 New York Avenue.

HEADACHE, DRUMHEAD RETRACTION AND VACUUM FORMATION.*

DR. EUGENE R. LEWIS, Los Angeles.

This paper presents my reasons for dissenting to certain tenets concerning commonplace upper respiratory problems; available proofs are offered and lack of other proof is clearly pointed out; no attempt is made to controvert moot points other than those specified concerning vacuum formation and drumhead retraction.

For years it has been held in Vienna that chronic adhesive process begins in utero, as mesenchyme of tubotympanic space is invaded by finger-like processes of embryonic ectoderm destined to epitheliate this space, and that fortuitous linear adhesions formed between these processes develop into transtympanic strands, which later gradually lose elasticity and undergo secondary changes recognized as "chronic adhesive process." Not because this postulate (whose quasifatalistic connotations include *futility of therapeutic effort* and *submission to the inevitable*) is inherently "unconstructive," and as such, obnoxious; not because I have been unable to find convincing evidence of both the fetal adhesions and the asserted transtympanic striae; but chiefly for the reason that I find in this type of adhesive process no features which are not as satisfactorily accounted for on the basis of *well recognized clinical*, and *more familiar histopathologic* findings, I am inclined to believe that many so-called "chronic adhesive processes" accordingly assumed to depend upon intrauterine etiology, are to be otherwise explained.

Intimately associated with this question is that of vacuum-formation. Certain drumhead retractions have been attributed to vacuum in tubotympanic space and certain headaches to vacuum in nasal sinuses, on the assumption that swellings, disturbed ventilations and air-absorptions lead to vacuum-formation (or some degree of negative pressure) in these cavities. In view of this etiologic and clinical association, these two problems are conjoined in this discussion. It

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is to be noted particularly that this concerns only what may come to exist in the body *under natural conditions.*

Certain observations have been interpreted as showing the existence of vacuum in these cavities; operators[1] have reported "sound of inrushing air" during drumhead (or opening the frontal sinus) incision. Even though incising a taut drumhead occasionally happens to produce sound which might be interpreted as "air rushing in" at the instant of myringotomy, this by no means proves existence of vacuum. Bryan and Alden[2] quote Sluder: "vacuum headache, established by closure of the frontal sinus—narrow nasofrontal duct may be temporarily closed by acute swelling. This will cause partial vacuum in the sinus." Nothing is stated to prove that this *does* cause "vacuum in the sinus." Just to the extent that local disturbances include means whereby air particles (and other particles) *may be moved* into some other space, some degree of air-absorption must be assumed; but to assume that air-absorption *evacuates* a cavity, in the sense of "producing vacuum," is erroneous. For particles to move, *force* is required to move them, and *space* for them to move into. In the absence of proof of its actual existence, the status of vacuum in working hypotheses must be established by examining the *structural and physical properties* of body cavities in connection with *requirements for vacuum-formation.*

Essentials to vacuum-formation include: rigid limiting walls, an evacuating mechanism and forces to operate it. None of these are evident in nasal or tympanic structures under natural conditions of health or disease. Sinus and tubotympanic walls are predominantly, but by no means entirely, *rigid; when their nonrigid* portions, together with their lining membranes and other tissue-contents become swollen, normal ventilation-openings are "blocked," and ensuing changes have been thought to lead to vacuum formation. Study reveals, however, that the existence of vacuum is *precluded by the very properties* which permit swelling and ventilation disturbances believed to produce vacuum. Logical approach to an understanding of actual conditions requires that the *forces* concerned in *general pressure changes* be reviewed.

Consider two *elastic* containers whose *cubic dimensions are equal when inflated* — the first with one volume, the second with two volumes of air at sea level; suitable petcock pressure gauges show 760 mm. Hg. pressure within the first, 1520 mm. Hg. within the second. On opening the petcocks, pressure within the first is found

to undergo no change, indicating that atmospheric pressure (760 mm. Hg.) is the *only force* acting upon its outer surface; pressure within the second is found to fall steadily as air escapes through the petcock. The size of this container is found to decrease and, as air escapes, its internal pressure continues to fall until the gauge registers 760 mm. Hg. As these containers are under the same atmospheric pressure it is evident that *additional forces* have been acting upon the outer surface of the second, whose shrinkage in size reveals the source and nature of these additional forces; the elastic material of the second has *conserved forces* by which it was inflated; these, together with that of atmospheric pressure, account for total external pressures sufficient to counter-balance 1520 mm. Hg. internal pressure. For this container to have *increased in size during inflation,* its internal air-pressure must have increased parri passu with its increasing exposure to external forces (atmospheric pressure and elastic tension of its walls). Both *during* and *after* its inflation, pressures exerted by internal forces have been counter-balanced by those exerted by external forces.

Negative pressures may be reviewed likewise by considering *rigid* containers, such as similarly equipped glass or metal spheres of equal volume, the first *filled,* the second *half-filled,* with air at sea level. Pressure within the first is found to be 760 mm. Hg., within the second, 380 mm. Hg.; on opening the petcocks pressure within the first is seen to undergo no change; it is seen to rise steadily within the second, as air enters through the petcock, until the gauge registers 760 mm. Hg. Atmospheric pressure *equals* pressure within the first; it *greatly exceeds* that within the second, which must therefore collapse unless *something additional* reinforces its internal air-pressure. Collapse is prevented by its wall, whose "material strength" (tensile and rigid properties) supplies what is needed to maintain balance between external and internal pressures.

In the course of many hundred examinations (under maximum illumination and magnification) inflation, pneumatic vibration and manipulation by Lucae probe have failed to *alter apparent shortening* of the hammer-handle; local or general movements of the drum-head may always be detected, but neither *rotation of the short process* or *apparent lengthening of hammer-handle* has been seen. On the assumption that retraction with apparent shortening owes its existence, even *partly,* to negative pressure in tubotympanic space, whatever amount of displacement of membrane and hammer *could be attributed to this cause* must be affected by such forced inflations

and gross disturbances of membrano-ossicular quiescence; and *some changes,* in the position of the manubrium and the relations of both short process and membrane, would have been discoverable.

Indrawing of the manubrium, such as is found in retracted drumhead with apparent shortening, *is caused* by contraction of the tensor muscle; irritations of the membrane and neighboring tissues produce definite spasm of this muscle. Tensor contraction, in itself, suffices to account for what has been attributed to negative air-pressure; when some force moves the manubrium inward, the head of the hammer is rotated into new relations with epitympanic tissues; sustained or repeated reflex irritations, spasmophile and exudative factors, combine to produce certain changes characteristic of "adhesive process." Organizing exudate initiates the fibrosis, calcification and chalk deposit found in suspensory ligament, tensor tendon and sheath, drumhead and mucoperiosteal tissues (with or without adventitious tags or adhesions), which constitute the chief pathologic characteristics of chronic adhesive process; similar tissue-changes are found elsewhere in such patients, varying in extent with the locus and habitus of individual reactivities and irritabilities, as conjunctival, nasal, aural, tracheobronchial, gastroenteric, pyelocystitic processes. With changing *textures, contours* and *bulk* of lining tissues and walls of epitympanic space, the action of the malleus ligaments and the working relations of the head of the hammer are affected, in the course of adaptations conforming to such changes. Even slight disturbance of the setting of the hammer-head causes marked displacement of the manubrium, and commensurately marked indrawing of the membrane. Incidental adhesions and contractures, in the course of years of recurring acute spastic and exudative episodes, ultimately bring the tip of the manubrium successively nearer to the promontorium, and throw the stapes into relief against the membrana tensa.

The conditions under discussion are *notably inconsistent* in their incidence and their clinical associations; extreme degrees of nasal and tympanic swelling exist without either headache or drumhead retraction; the latter is found frequently in the absence of history or signs of tympanic trouble and, on the other hand, it is absent in a large number of cases which *do* show tympanic trouble.

Drumhead retractions with or without adventitious cicatrization, fibroses, calcifications, etc., have been variously attributed to "catarrhal," "fibrosing," "cicatricial" and "congenital" changes. It is true that swellings tend to produce profound ventilation disturbances in

the cavities under discussion; it is also true that *incarcerated air* undergoes absorption, which means that it, or *some* of it (oxygen, nitrogen, carbon dioxide, etc.) *goes elsewhere*, according to laws determining the behavior of fluids under pressure. Nevertheless, without entering into details concerning diffusion and osmotic pressure, it is clear that neither tympanic or sinus structures meet requirements for vacuum-formation; and this theory must, therefore, be discarded. The adhesive conditions attributed to prenatal tissue-changes will bear further study and consideration, with respect to alternative factors of etiology and more familiar concepts of pathogenesis.

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1154 Roosevelt Building.

CEREBROSPINAL RHINORRHEA.

Dr. Arthur I. Weil and Dr. David R. Womack,
New Orleans.

This case of cerebrospinal rhinorrhea is reported because it is the authors' belief that the condition is much more common than the recorded cases would indicate and it is the earnest hope that interest will be stimulated to the extent that cases will be reported as found. In this way authentic statistics will become available as to the prevalence and character of this condition. Cases are recorded as the result of surgical procedures and we must condone our confreres on their courage and assure them it is no reflection on surgical skill. Only by a complete report of the cases will our statistics be of any value.

In the past three years three cases have come under the observation of one of us and in conversation with other men it would seem they see a number of cases; all of which would belie the literature, where we find some seventy-three cases recorded—and twenty-one of these were collected prior to 1899.

St. Clair Thomson published a monograph in 1899 on "The Cerebrospinal Fluid; Its Spontaneous Escape from the Nose," in which he records *twenty-one* cases (not *twenty*, as is recorded by some who quote him but evidently have not read his book) of varying types. He very exhaustively describes the condition and its recognition.

Roth reports a case of spontaneous cerebrospinal otorrhea (*Archiv. Otolaryngol.*, 13:3, p. 439, Mar., 1931) as "the first report of a spontaneous cerebrospinal otorrhea," but in St. Clair Thomson's monograph, on page 6, a case is recorded as reported by Escat, of Toulouse (*Archiv. Internat. de Laryngol.*, Tome, X, No. 6, 1897), of spontaneous cerebrospinal otorrhea. So even cerebrospinal otorrhea is not so rare, having been recorded over thirty-five years ago.

Thomson says, "as in so many other affections at first recorded as 'rare,' it may be found that cerebrospinal rhinorrhea is only rare from not having been carefully looked for."

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Johnston, in 1926, gave a very excellent survey of the literature with a report of twenty-one cases since the time of St. Clair Thomson's work—forty-two cases in all up to 1926. He calls it a "rare" condition.

Donnelly, in September last year, records sixty-nine cases and reports two more, which makes seventy-one cases. Since this date Fox has reported a case which, together with our case, makes a total to date of seventy-three cases of cerebrospinal rhinorrhea.

The condition is so thoroughly covered in the literature that it would be useless to recount its etiology, history, diagnosis, prognosis and treatment.

Case Report: The patient, a colored female, age 40 years, presented herself to the Touro Infirmary Clinic, Department of Otolaryngology, on July 21, 1932. Her complaint at this time was "water dripping from her nose." Her family history revealed nothing of note except that her family have been marked for their longevity.

The past history revealed the usual childhood diseases. Never has the patient had any injury, no history of external violence at all. She has had no serious illnesses. Six years ago patient had a "nose operation" at another hospital. Recovery from this was complete in four weeks and patient has had no complaints from that time until the beginning of her present trouble, a period of five years and three months. Communication with the other hospital brought the following answer: "Was first admitted to our outdoor clinic on April 8, 1927. The diagnosis at that time was chronic epipharyngitis. An X-ray showed an opacity of the sphenoids. Left sphenoidectomy was performed on April 30, 1927, under local anesthesia."

Examination reveals a well developed and nourished colored woman, age 40 years. There is a constant drip from the left nostril when the head is held forward. After counting the drops on several occasions the average is about ten drops per minute. The rate varies with exertion and straining, going up as high as fifteen drops per minute. There are no symptoms referable to the eyes, no headaches, dizziness, nystagmus, etc. It is significant that the patient has had no headaches and that drop is continuous. The discharge runs in the throat when the head is on pillow. This discharge is clear and watery and does not stiffen when it dries on handkerchiefs, linens or clothing.

Examination of the nose shows a mass on the left side which is glistening, pearly gray and is some 1 x 1.5 cm. in dimensions and

projecting seemingly from the left sphenoid region. This mass is rather firm and nonpulsating. The mass is moist and shiny; and somewhat resembles a polyp. On lowering the patient's head almost to the floor in extreme Trendelenburg position there is no change in the size or position of the mass. It was considered inadvisable to disturb this mass too much as it was thought to be a hernia of the brain substance. The middle turbinate on this left side, of course, has been removed. The right side is intact. Otherwise the nose is negative. The fluid was clear and transparent. It was collected and sent to the laboratory and the report returned was as follows:

"Dextrose, 52.5 mgm. per 100 cc.; NaCl., 655 mgm. per 100 cc.; evidently this is cerebrospinal fluid.—Dr. John A. Lanford."

An X-ray of the sinuses showed: "Both maxillary antra are showing considerable clouding and the ethmoid cells on the right are likewise very cloudy. Aside from this, however, no evidence of disease can be detected. It is impossible to see any abnormal opening in the skull, so that radiographically it is impossible to determine the point from which the cerebrospinal fluid is coming."

The blood Wassermann was negative.

We had this patient under observation about three weeks, during which time her condition was unchanged and she was in apparently her normal good health. She suddenly stopped her visits to the clinic and after much searching we found her brother, who told us the patient "had died two weeks ago." She was attended by a colored physician of our city, who was communicated with and thought patient had a "cerebral hemorrhage," but on questioning he admitted the patient had some fever ("an even hundred") and pathological reflexes. Her blood pressure was 160 the day before her death. It is perfectly possible that the cause of death was a terminal meningitis but this cannot be proven as no autopsy was obtained.

This patient never presented any eye symptoms. We had referred her to the eye department on her last visit to our clinic but she did not go that day and, unfortunately, she died before she could carry out our instructions; thus we have no eye examination to report.

There are a number of thoughts brought up by this case. Did the previous nasal operation cause this herniation of the brain and so cause the rhinorrhea? Why should the interval between operation and rhinorrhea be so long — over five years?

This case is presented to encourage other case reports and a detailed description of the disease will be omitted. The bibliography

attached is ample and anyone so desiring may pursue the matter more fully.

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1128 Maison Blanche Building.

COMMUNICATION.

Nashville, Tennessee,
July 31, 1933.

To the Editor,
THE LARYNGOSCOPE,
St. Louis.

Dear Sir:

I have just read an article in the July number of THE LARYNGOSCOPE entitled, "A New Mouth-Gag with Interchangeable and Adjustable Tongue Depressors and Anesthetizing Tube," by Dr. Walter Stupka, of Neustadt, Austria. It is the same gag in principle that I invented and showed before the Ear, Nose and Throat Section of the A. M. A. at the Atlantic City meeting in 1912.

It undertakes to do what my gag does but in a clumsier and more inefficient manner. In fact he fails to stress the point for which I make the greatest claim, namely opening the larynx and aiding respiration.

There were numerous mouth-gags with tongue depressors attached before mine but they all had the same drawback, namely the tongue depressor was attached in the middle and consequently crowded the base of the tongue down onto the larynx, thus impeding respiration instead of aiding it. By having the tongue depressor attached on the side, my gag allows the tongue to rise between the teeth and the depressor. The tongue is drawn out of the mouth and curved tongue depressor is placed against the base of the tongue and adjusted. When the depressor is properly adjusted it exerts a lifting force at the base of the tongue and opens up the larynx, promoting free respiration. When the depressor is properly adjusted one can often look directly into the larynx, as in suspension laryngoscopy. Indeed the late Dr. Robert C. Lynch said to me on more than one occasion that he could suspend a patient with my gag. Dr. Stupka appears to have overlooked this point as his tongue depressor appears to be attached in the middle.

My gag is of the self-retaining type and consequently the upper and lower bites are always parallel. My gag is of stronger, sturdier

construction as the scissors opening in the instrument will tend to wear with use and get rickety. My gag has been in constant use since 1911 and is as good as ever.

I notice the instrument is made by Carl Reiner. While in Vienna in 1913 working in Marshik's Clinic in the Algemeiner Krankenhaus I showed my instruments to Reiner, who copied them. He afterwards put out an exact copy of my tonsil clamp under the name of "Marshik's Tonsil Clamp." It was marketed in this country by E. B. Meyrowitz & Co. I mention this to show that while Dr. Stupka may not be familiar with my gag, Reiner is thoroughly familiar with it and all the principles involved.

The attachment of the anesthetic tube is nothing new. I thought of adding it, but it is really a drawback instead of an aid. It is more convenient and simpler to have the vapor delivered through a rubber tube with a metal hook on the end which can be placed in the corner of the mouth. It is out of the way and can be instantly removed or replaced without having to be attached or detached.

Yours truly,

M. M. Cullom, M.D.

Suite 1119-24, Bennie Dillon Building.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The thirty-eighth annual convention of the American Academy of Ophthalmology and Otolaryngology will convene in Boston. Sept. 16 to 22, 1933. Hotel Statler has been selected as headquarters. In keeping with the general progress of things, the program for this meeting is more elaborate than those that have preceded.

The American Board of Otolaryngology will hold an examination in Boston, Saturday, Sept. 16, at the Massachusetts Eye and Ear Infirmary. Those desiring appointments should communicate with the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb.

The American Board for Ophthalmic examinations will hold an examination on Tuesday, Sept. 19, at the Massachusetts Eye and Ear Infirmary. Information may be secured from the Secretary, Dr. W. H. Wilder, 122 South Michigan avenue, Chicago, Ill.

RESEARCH STUDY CLUB OF LOS ANGELES.

The Research Study Club of Los Angeles will present its third mid-winter course January 15 to January 26, inclusive, 1934. Guest lecturers in Otolaryngology will include Dr. John Barnhill, of Miami, Florida; Dr. E. C. Sewall, of San Francisco, California, and Dr. Harry L. Baum, of Denver, Colorado. Dr. Barnhill will give a course in dissection and surgery on the cadaver. This was the most popular feature of the course last year.

The guest lecturers in Ophthalmology will be Professor A. Elschnig, of the German University Eye Clinic, Prague, Czecho-Slovakia, and Dr. Hans Barkan, of San Francisco.

The lectures have been so arranged this year that participants in the course may take all ophthalmological and otolaryngological subjects without overlappage.

For further information and a detailed program, address the secretary, Dr. Don Dryer, 2007 Wilshire Blvd., Los Angeles, California.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION OF OTO-LARYNGOLOGY.

Meeting of April 14, 1933.

Dr. H. J. Rothschild, President, presiding.

DR. W. P. LARSON (University of Minnesota) (by invitation) gave a talk on Bacterial Allergy.

DISCUSSION.

DR. J. A. WATSON (Minneapolis) said he had been much interested for some time in the whole question of allergy, anaphylaxis and atopy. He believed that allergy is a peculiar, always inherited, and not understood constitutional condition which makes an individual much more likely to develop sensitivity to foreign protein than an individual who has no such inheritance back of him, and he believed that bacterial proteins were in the same category as other proteins in this regard. How, otherwise, he asked, can the fact be explained that a certain percentage of people with focal infections will develop allergic symptoms, while the balance will not? Why should one child, for instance, develop asthma with sinus trouble, and another not? Undoubtedly because in the one case the child has an allergic background which makes him more easily sensitized to bacterial proteins in common usually with many other proteins. At least Dr. Watson felt that he had been able by observation of a sufficient number of cases to determine the presence of sensitization to other proteins in a considerable number of cases of bacterial allergy, or, if not, to establish an allergic history in the family.

DR. LARSON said he had no doubt but what family history is extremely important. It is known that patients who have hay fever are irritated not only by the pollen with which they are sensitized, but by many other inert substances as well. There is also an individual difference toward these substances. In experimental work, a group of guinea pigs may be sensitized with egg white and then when they are brought before a class perhaps one will find only about half of those pigs will respond or react. So there are individual differences in the way they respond. Not as much is known about the allergic state in the human as in animals, because experiments cannot be carried out so well on the human. But Dr. Larson said that in his study of allergic conditions the family history would be one of the important factors.

DR. W. E. CAMP (Minneapolis) said this discussion certainly had been very interesting and has cleared up many things which are seen clinically. He wished to ask one or two questions. Quite frequently the oculist sees cases of supposedly iritis that are due to focal infection — usually a tooth. After the extraction of the tooth the iritis subsides. Could that be considered an allergic reaction or a definite infection through the blood stream? The second question is that of adults who develop hay fever who have never before had it. One would think if that were a congenital condition it would show up in early life. Frequently one sees adults who suddenly develop hay fever and have never shown any indications of it before.

DR. LARSON said, in regard to Dr. Camp's first question as to whether the iritis was a case of atopy or allergy or infection, he supposed it might be both, but the only way one could get a better idea would be to isolate the organism and do a skin test.

In regard to cases of hay fever developing late in life, Dr. Larson said most cases of hay fever will develop in the first ten years of life. However, he had seen two cases which developed after the age of 30; it is quite rare, but it does occur. Dr. Larson said he did not know that any one has tested the

blood of these patients by passive transfer, and he did not know how long these patients had carried these reagins in the blood before they had symptoms. It has been shown in some children that they will carry the reagins two years before. One would expect the symptoms to appear as soon as the reagins appear, but they do not.

DR. PAUL BERRISFORD (St. Paul) cited a case which, for the moment, gave him much anxiety. He had treated a young lady during two seasons with 15 graduated doses of Lederle's pollen antigen. In the third season he had just administered the 14th dose when, within less than a minute, the patient became suddenly and violently ill, so much so that she appeared on the point of death. There was marked cyanosis, marked shallowness and rapidity of breathing, weak pulse, puffiness of the face, especially of the eyelids, and a generalized cutis anserina ("goose-flesh"). Breathing was so difficult that the patient found it necessary to sit up to get the necessary air.

In the course of a few minutes the "attack" had completely disappeared and the patient left the office apparently no worse for her experience.

Dr. Berrisford thought this affair very peculiar in view of the fact that this phenomenon arose during the 14th dose and also in the light of his past experience with the case.

He asked for Dr. Larson's explanation of the occurrence of this apparent case of anaphylactic shock. Could it have been anticipated and how guarded against?

DR. LARSON said he had seen one case just like the case cited by Dr. Berrisford. The patient was a farmer boy who was a very acute hay fever subject. He, also, had received the 14th dose and shortly afterward became so ill that he had to go to bed. He got over that and went back for the 15th dose. He collapsed on the doctor's table, but the doctor brought him out. Dr. Larson said the only explanation he could give for that is that the first fourteen "shots" did not sensitize and make the patient tolerant to the last dose. He said it was just cases like this that make him a little pessimistic about the bacterial toxins.

DR. ROTHSCHILD (St. Paul) said he was sure the members of the Academy all felt this had been a very instructive presentation, and he wished to thank Dr. Larson.

DR. GEORGE S. MONSON (St. Paul) (by invitation) gave a talk on "The Restoration of Occlusion of the Teeth as an Aid in the Restoration of Impaired Hearing and Deglutition." This was illustrated with lantern slides, blackboard charts and specimens.

DISCUSSION.

DR. ROTHSCHILD (St. Paul) asked Dr. Monson if any observations had been made in regard to the pressure of the condyle on the tympanic twig of the internal maxillary artery as it passes through the glaserian fissure.

DR. MONSON said he had not been able to determine that, but in cases that have had pronounced tic douloureux there was a closure and the impingement could be felt, and as soon as that was opened up the patient lost the tic douloureux.

DR. J. A. WATSON (Minneapolis) said he would like to ask whether any series of observations had yet been made which would tend to show that hearing is impaired more frequently in those cases where there is malocclusion than in patients with normal dental arches, or to show that in any considerable number of cases the hearing is improved after the malocclusion has been corrected. He doubts the theory of pressure on the floor of the external auditory canal having anything to do with impairment of the hearing, in view of the fact that the external auditory canal may be almost entirely occluded, as, for instance, by wax, without any change in the hearing so long as there

is any passage at all left open. Pressure by the condyle of the jaw in the glenoid fossa or glaserian fissure might affect the hearing through interference with the circulation or the nerves.

DR. MONSON said he was under the impression at first that there was a relationship, for one could have trauma which would interfere with hearing, but he had found they got no results in the patients until the function of deglutition had been restored. These patients had become indolent in swallowing. The Eustachian tube, when crowded by structures of the oral cavity, did not drain. Practically all those cases had become indolent in swallowing. It is quite a common observation that deaf people will keep their jaws open in listening. Dr. Monson said in his own case he had to resort to occlusal splints on his teeth. He first took audiometer readings, then slipped the splints off for three days. The following day he took the reading and there was a drop in hearing, and the ringing in his ears which he had had two years previously began again.

DR. ROTHSCHILD stated it seemed to him that Dr. Monson had brought out very well that dental malocclusion may produce malposition of the muscles of deglutition and thereby produce pressure on the Eustachian tube, with its possible deleterious effect on the hearing.

DR. HORACE NEWHART (Minneapolis) expressed appreciation for this very fine presentation of Dr. Monson. He asked if the audiograms recently made showed the hearing loss particularly in the high or low tones.

DR. MONSON replied: "In the higher tones."

DR. NEWHART expressed the opinion that this rather contradicts the supposition that the hearing loss is due to an involvement affecting primarily the Eustachian tube.

DR. C. E. CONNOR (St. Paul) felt that all the members would welcome any new approach to the problems of the hard of hearing. He felt that Dr. Monson's work would be more convincing if the hearing had been thoroughly tested before and after the work was done. Dr. Connor felt that the word of the patient as to what has been accomplished in any line of therapy is not always reliable, as the patients are inclined to be optimistic and to feel for a time that they are being benefited. He said he could conceive how the hearing could be impaired if the Eustachian tube were impinged on, but it was difficult to see how pressure on the bony external canal could cause it. He felt this was a very interesting point of view and that Dr. Monson should follow the work up with some complete functional studies.

DR. MONSON stated that the bulk of this work was done prior to the perfection of the audiometer. They did use tone instruments to give a comparison.

He said he was appearing tonight before this body to enlist the cooperation of this group of specialists in order to put the work on a more scientific basis. He said it had come to him not as an objective, but as a result.

DR. ROTHSCHILD thanked Dr. Monson and stated he felt this had been a very interesting topic and he hoped Dr. Monson would be able to present further studies along this line at some future time.